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VOL. I.—14TH YEAR. SYDNEY: SATURDAY, JUNE 18, 1927. No. 25.

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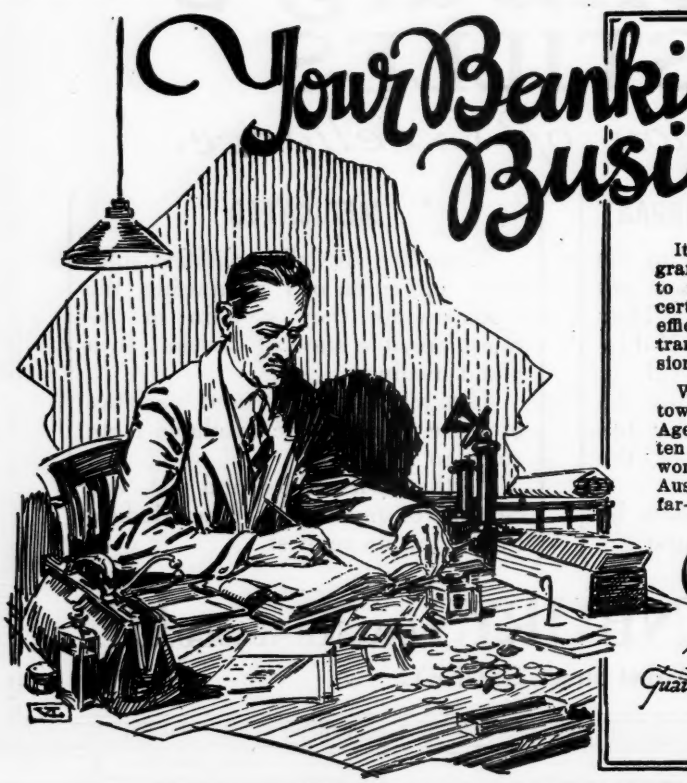

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RODENT ULCER.[1]

By E. H. MOLESWORTH, M.B., Ch.M. (Sydney),
Honorary Dermatologist, Royal Prince Alfred Hospital, Camperdown, Sydney; Lecturer on Diseases of the Skin, The University of Sydney.

INTRODUCTION.

THE original object of the work described in this paper was to obtain definite evidence, if possible, of the tissue of origin of rodent tumours and of the relation of the latter to squamous epithelioma.

After a considerable amount of work the problem of causation attracted a great deal of interest on account of the definitely heavier incidence of rodent disease in Australia as compared with England and because the reasons usually advanced to explain its occurrence could not be made to fit in with clinical and pathological facts collected.

Some evidence bearing upon the second line of inquiry was not recorded when the earlier specimens, excised for the purpose of elucidating the tissue of origin, were obtained. But it was fortunately possible to correct this omission by subsequent observations of other cases in plenty.

Exceptions, real and apparent, occurred, of course, but none were entirely inexplicable by the hypothesis advanced as a result of the examination of the evidence made available on the subject.

The number of specimens examined may be gauged by consideration of the fact that after rejecting those with no definite or special value, over a hundred were kept and a final selection of about forty constitute the pathological material of this paper. As a great many of these were examined in serial section, it can be understood that this selection and the subsequent interpretation occasioned an enormous amount of tedious labour. Therefore the failure to pursue all avenues of inquiry must be excused for the reason that it is so difficult to avoid being dragged into all sorts of by-paths.

In respect to any errors of interpretation the same indulgence might be allowed to a man who, though much interested in the pathology of diseases of the skin, has been mainly active on the clinical side.

Specimens were sent by colleagues who were interested in the inquiry either for scientific or personal reasons, from distant parts of Australia, as well as from Sydney and to all of these a deep debt of gratitude is owing for the help and encouragement they afforded in the course of the work which has extended over a long period of years.

The bulk of the material was derived from the Out-Patient Department of the Royal Prince Alfred Hospital, Sydney, and there was available for the purposes of comparison the resources of the Pathological Department of the University of Sydney and sections sent by Dr. Whitfield, of King's College, London, as well as some obtained years ago in the laboratory of Professor Jadassohn then at Berne.

[1] Submitted as a thesis for the degree of doctor of medicine at the University of Sydney.
This paper has been offered to the editor of *Urologic and Cutaneous Review* for simultaneous publication.

The pathological work was performed under the supervision and in the laboratory of Dr. Welsh, Professor of Pathology of the University of Sydney, whose guidance, kindness and patience in the matter are greatly appreciated.

It is not unlikely that the findings on the matter of causation will create opposition, because to some they will appear novel, to others unconvincing. But it is felt that in Australia at least, the home of rodent and allied conditions, they will occasion little surprise and less opposition. The fate of the conclusions can be confidently left to others, since they are capable of corroboration though not without difficulty, patience and careful discrimination in noting clinical data in a large series of cases on the lines indicated in the body of the paper.

The conclusions as to origin are upon much more conventional and generally accepted lines, though here too it is probable that in some quarters they will meet with disagreement.

The years of consideration of all aspects of the subject matter of the paper have left me confident of the general soundness of the findings on both questions and I would welcome any checking which might be undertaken.

My thanks are due to the members of the staff of the Pathological Laboratory of the Sydney University who performed the tedious and to them uninteresting labour of fixing, cutting, staining and mounting the great number of sections used in the series with never failing cheerfulness, readiness and care.

PART I.

This part consists simply of statements of fact and clinical observation. These observations have extended over a period of ten years or more and have been based on a number of patients amounting to many hundreds seen in the Royal Prince Alfred Hospital, Sydney, and in private practice. So there has been ample opportunity for checking and discussing them and this has been freely done.

Clinical Observations.

General: On the Frequency of Rodent Disease and Squamous Epithelioma and on the Rarity of Lupus Vulgaris.

In the pursuit of the study of diseases of the skin along clinical and pathological lines there becomes obvious a very remarkable difference between the respective incidence rates of two important diseases, when observed in the same race domiciled in England on the one hand and in Australia on the other. These differences are:

1. The much greater relative frequency of *lupus vulgaris* in particular and of tuberculous diseases of the skin generally in the home country than in Australia where lupus is exceedingly rare and is noted only in people who have come here with the disease already developed.

2. The disproportionate frequence of rodent ulcer and squamous epithelioma of the skin in Australia as compared with the incidence of the same diseases in the same race living in England, Scotland and Wales and even in North America (with the exception of California and States of similar climates).

There can be no gainsaying these observations. The facts have been frequently noted and comment upon them freely made, but no correlation between the two has been drawn.

In the course of sixteen years' work in the Skin Department of the Royal Prince Alfred Hospital, Sydney, with as large an attendance of patients as most London hospitals and larger than some, there occurs an average of a little more than one patient with *lupus vulgaris per annum*. If to these are added the extremely rare cases which come in private practice a total of twenty-five would be a liberal estimate of the number of such patients coming under my notice in sixteen years in Sydney. Of these one only developed the disease in Australia (in a coal mining town), all the rest having arrived in the country with the disease already established, nearly all from Europe, one from New Zealand and one from America.

On the other hand rodent ulcer, squamous epithelioma of the skin and pre-rodent or pre-epitheliomatous lesions are so common as to provide the bread and butter work of the dermatologist in Australia. Literally many hundreds of patients are treated *per annum* in the hospitals and many more in private practice in Sydney.

Particular Observations: Of a Clinical Nature Relating to Rodent and Squamous Epithelioma.

Localization.—The almost invariable development of rodent tumour on the face and neck and occasionally on the upper part of the chest is well known, but none the less remarkable. Its especial predilection for the middle third of the face, which is an area occupied by the most active sebaceous glands, is also commonplace knowledge. The great rarity of rodent on other parts of the body is shown by the fact that on three occasions only among many hundreds and possibly thousands of patients have I seen rodent tumours or tumours indistinguishable from the latter by histopathological examination occurring on other portions of the body. Two of these lesions occurred on the back and one on the thigh: (i.) Mrs. M., a private patient, (ii.) Dr. Paul's patient in the Sydney Hospital and (iii.) Dr. Hamilton's patient in the Sydney Hospital. In the second and third the condition was Bowen's epithelioma.

The first of these was ulcerated, the other two were fine, pearly edged lesions with atrophically scarred centres, but no ulceration. On section definite basal celled carcinoma was recognized.

The occurrence of pre-rodent lesions in the shape of keratoses is not so generally admitted as is the existence of pre-epitheliomatous keratoses, but the existence of these formations is readily demonstrable in Australia by a combination of clinical and histopathological methods to be referred to later. Moreover, there sometimes occur in addition to keratoses (hypertrophic lesions) other signs, such as pigmentation, patchy atrophy of the skin and even telangiectases, a picture very reminiscent of a moderate degree of chronic radio-dermatitis. This is common in association with squamous epithelioma of the skin, but it has also been definitely and not infrequently noted in cases of rodent ulcer.

The pre-rodent keratosis can generally be distinguished fairly readily from that preceding squamous epithelioma. It is finer and flatter and more like that of *lupus erythematosus* in texture than the coarse, heaped-up lesion approximating in appearance to the cutaneous horn which seems to develope almost invariably into squamous epithelioma.

The development of a tumour under the pre-rodent keratosis in a manner somewhat similar to that of squamous epithelioma under a cutaneous horn is the next observed stage of evolution of rodent disease. This clinical observation can be checked by examination of numbers of the sections to be described in the part devoted to histopathology. Indeed it was by excision of flat keratotic lesions with slight suspicion of underlying infiltration that some of the best examples of early rodent disease to be shown later were obtained.

The result of the formation of this little tumour is the stretching and consequent thinning and atrophy of the covering epithelium. This determines the shedding of the keratotic cap and reveals the small pearly tumour universally recognized as a true rodent formation.

At this stage a divergence in the lines of development occurs, but whether the line followed is inherent to the particular tumour or determined by conditions such as continued irritation or other causes, it is impossible to say. But the fact remains that some of these little tumours tend to spread peripherally while crusting and scarring, or cicatrizing without crusting at the centre, thus forming the rodent tumour of superficial cicatrizing type; or on the other hand it may ulcerate early though generally leaving at the periphery an unulcerated, pearly ring of infiltration which generally serves to distinguish from squamous epithelioma with its everted edge. This last type tends to involve deeper tissues, the former rarely penetrates deeper than the true skin, not as a rule involving deep fascia and subjacent muscles until late. But even the deep infiltrating rodent growth is slow of progress, causes no pain, frequently has long periods of quiescence and only very gradually, sometimes only in the course of several years tends to produce alarm in the minds of some patients.

On occasions, but in my experience rarely, rapid growth occurs and still more rarely fungation.

As examples of the extraordinary variation in development and of chronicity of course of the ulcerating type of rodent tumour, the two following histories are interesting.

Mrs. M., about fifty years of age, appeared at Saint Vincent's Hospital, Sydney, with a rodent tumour which had extended superficially by ulceration until it had involved almost the whole of the left side of face and scalp, had totally destroyed the left pinna and had invaded the middle ear by way of the external auditory meatus. On inquiry the patient admitted that the disease had been in existence for fifteen years and explained that she thought that because it did not hurt, except when knocked, it

could not be serious and she expected it to heal spontaneously at any time.

Mrs. W., who presented herself at the Royal Prince Alfred Hospital, Sydney, with two rodent tumours, one on the left side of the forehead, the other on the left posterior part of the neck just below the mastoid. The history was that the lesion on the forehead had existed for ten years and that on the neck for eight years. Except for the length of history the clinical evidence as far as concerned the lesion on the forehead was much more in favour of a diagnosis of deep penetrating gumma than of rodent ulcer. This ulcer had the appearance of a trephine hole about the diameter of a two shilling piece, bored straight back through the frontal bone, the base of the ulcer being the *dura mater*. However, on section the ulcer proved to be of ordinary rodent formation.

Observation of Multiplicity of Rodent Tumours.

The multiplicity of lesions in cases of rodent disease is even challenged by some European authorities, but it is quite commonplace in Australia to find cases of multiple rodent tumours. Two or three frequently and seven or eight with intervening innumerable keratoses have occasionally been observed. A still more frequent occurrence is the appearance of a patient with one well developed and obvious rodent tumour associated with numerous keratoses which are here and there accompanied by a little subjacent thickening and infiltration.

As examples of multiple rodent tumours can be quoted the second case described above and also that of Captain H., who after having had two lesions excised presented himself with seven rodent tumours of the superficial cicatrizing type on the face and neck. Numerous other instances could be quoted if it were necessary to further illustrate this point.

Occurrence of Tumours Simulating Rodent Tumour.

Beside Brooke's tumour (benign cystic epithelioma) and tricho-epithelioma, which in my experience are excessively rare in Australia, there is to be noted a small growth which appears on the face, generally in the neighbourhood of the naso-labial fold. It is single, is not preceded by any keratosis, rises steeply from the surrounding skin in a dome-shaped button which is almost translucent, and is sometimes slightly lobulated like a white raspberry. It is semi-cystic to the touch in distinction to the firm solidity of the pearly rodent tumour. It is exceedingly slow of growth, taking up to ten years to reach the size of a boot-button and even then only sometimes shows a tendency to ulcerate. A specimen of this tumour (SU2722) occurs in the present series; others were diagnosed clinically, but excision was not permitted. So in the absence of any example of Brooke's benign cystic epithelioma and only one doubtful specimen of tricho-epithelioma, it must be considered commoner than either in Australia at least. This is one variety of the tumour to which Macleod[6] refers as the cystic rodent growth. Though this growth can be easily distinguished from rodent tumour by clinical history and signs, it is generally diagnosed as a form of rodent disease and as it yields readily enough to radiation treatment, like rodent ulcer, it rarely comes to biopsy. This may explain why it has not already been extricated from the rodent group, even if not from the epitheliomata.

In histopathological characters, too, it bears fairly close resemblances to rodent tumour, as will be detailed later, so it is not difficult to understand why its individuality has been overlooked.

An unobtrustive, unimportant little tumour it is nevertheless interesting and should not remain undistinguished from rodent disease. It is possible that this tumour is responsible for the recurrent suggestion that rodent tumour is an endothelioma, a theory which is incompatible with clinical and I think pathological evidence.

Observations upon the Type of Skin Subject to Rodent Ulcer.

In the course of the collection of the large numbers of specimens examined for this study, it was forced upon my notice that certain types of skin were met with far more commonly than they occur in the community generally. This applies also to patients suffering from squamous epithelioma, but none the less certainly to those with rodent disease. The type referred to is that characterized by a very fair skin. By subsequent checking this was found to be correct, as the following series of cases demonstrates. First noted was the large proportion of red or sandy haired patients. But the colour of the hair was subsequently found to be undependable as a criterion. For example, brown hair with blue or grey eyes was found very frequently with a very fair skin. In fact the colour blue or blue-grey of the eyes was much more often associated with such a skin than was any particular shade of hair. But perhaps the commonest accompaniment of rodent disease is a tendency on the part of the skin to freckle, a characteristic which was sometimes, though rarely, found even in company with brown eyes.

Another observation was the disproportionate number of Irish and Scottish patients. This is probably explained by the tendency of Scotch and Irish blood to produce red or sandy hair and of Irish to produce that combination of brown hair, blue or grey eyes, with a very fair, clear skin (the Irish beauty type).

The fair Scandinavian and north German type was also found to be affected with rodent disease in numbers quite out of proportion to their representation in the population generally.

The English come next, but seeing that English form the largest section of the community, their representation in the rodent disease figures is proportionally very much smaller than is the case with the Scottish and Irish sections. Some allowance must be made for the facts that though fair hair and blue eyes are largely represented in English stock, there is also a number as large or perhaps even larger of folk of a darker complexion and even among the fairer individuals one finds often a much greater capacity for even browning and much less tendency to freckle than is the case in the brown haired Irish type mentioned above.

People with brown eyes and olive skins very rarely come into the rodent disease list. I have never seen an Italian with rodent disease nor could Dr. Fiaschi

with his large experience recall such an occurrence in his fifty years of practice in Australia.

No instance of a Chinaman or coloured patient suffering from rodent ulcer could be found, although many of such people pursue outdoor occupations, generally as market gardeners.

Observations on the Kind of Occupation which Seemed to Predispose to Rodent Disease.

Coincident with the realization of the predominant type of complexion represented in patients suffering from rodent disease came the observation that a very large proportion of such patients had passed several years or the whole of their lives in the country or at least had followed an outdoor occupation. In the case of the few patients whose actual livelihood was gained by indoor occupations, outdoor habits, hobbies and sports were nearly always admitted, as indeed was obvious from signs of exposure present on the skin. The women patients were not only of susceptible types of complexion, but in nearly all cases were of outdoor habits and showed signs of exposure.

This observation is, of course, not new, as is illustrated in one of the German names for rodent tumour *Seemanshaut Karzinom* and in Darier's description of *Cancer des Cultivateurs,* but the extent of its application to rodent causation has not been realized.

By far the great majority of cases of rodent disease occurs in individuals over thirty-five years of age, but occasionally in this country one finds younger patients in a big out-patient department, such as that of the Royal Prince Alfred Hospital. The youngest who came under my personal notice, was a youth of twenty and a more typical case of susceptible type of skin which showed every mark of exposure, it would be impossible to imagine. He was a sandy haired, freckled country boy of Irish-Scotch descent, who had been much exposed from babyhood and who had a tumour that proved to be a definite rodent on pathological examination of a snipping. There were multiple lesions which promptly healed under X ray treatment.

The same type of skin can be noted, the same history of abnormal exposure can be obtained in all such cases occurring in patients under thirty-five years of age. I have never yet seen an exception to this rule. Even in patients over thirty-five years of age, as will be seen in the following analysis of a series of thirty-seven successsive patients seen in my private practice, the types are found in fairly accurate order of susceptibility, the occupations will be found to be almost invariably outdoor and a disproportionate incidence in the Scotch, Irish or partly Scotch or Irish inhabitants is to be observed.

Of the thirty-seven patients twenty-three were men and fourteen were women; there were nine English, eight Scots, nine Irish, four of mixed Scotch and Irish blood, five of Scotch or Irish blood mixed with English and two Germans. This gives a total of twenty-six out of thirty-seven with Scotch or Irish blood, a number entirely out of proportion to the representation of these stocks in the community, as also holds in the case of the two Germans. This is not a freak proportion, but is fairly characteristic of the rodent disease incidence. Having no Scotch or Irish blood in my veins and being neither a Presbyterian nor a Catholic, it cannot have been that this proportion was due to racial or religious preference on the part of the patients consulting me.

The types of complexion were all noted. These were found to be:

Definitely red or sandy	6
Fair to sandy	11
Very fair	6
Fair	5
Brown hair, blue eyes, fair skin	7
Rather dark	2
Total	37

Of the two marked as rather dark, one is noted as showing freckling. This is to say that thirty-five out of thirty-seven were of distinctly fair type.

Next the occupations of the men were:

Farmers and graziers	9
Other occupations entirely outdoor	13
Indoor occupation (accountant)	1
Total	23

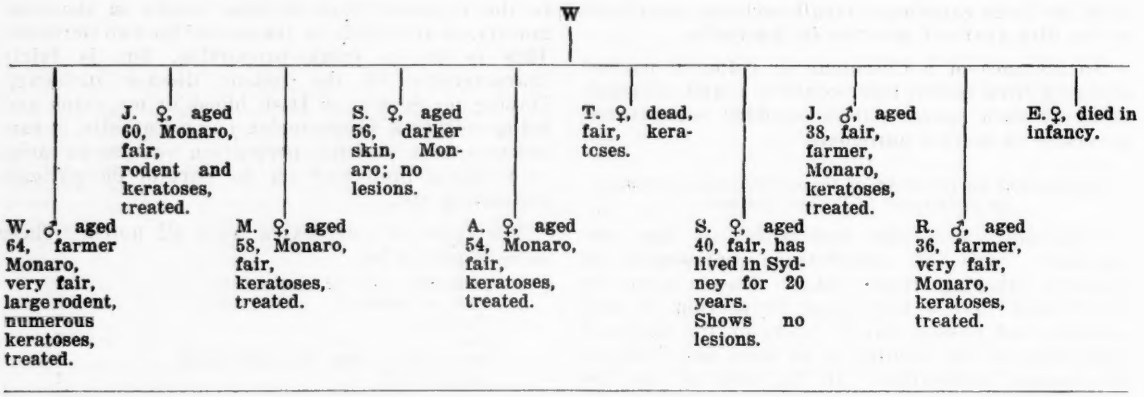
The accountant bore obvious marks of exposure and had for many years been an ardent amateur fisherman and gardener. Even eliminating this patient, twenty-two out of twenty-three were outdoor workers.

Of the fourteen women eight were country women who admitted to a lot of exposure and manifested obvious signs of it, two were country schoolmistresses and both showed many exposure effects, two were entirely outdoor workers, one was occupied only with household duties, but for many years had been a keen golfer, one was occupied with domestic matters and gave no history of undue exposure, making a total of fourteen.

Thus in all the women but one definite influence of exposure could be traced and all were of susceptible types.

Taking the whole thirty-seven, however, no patient in this series was under forty years of age and only ten were under fifty. So it appears that about forty years' of exposure is necessary to produce definite rodent disease formation, except in unusually susceptible folk. The full development into rodent tumour, however, may not reveal itself clearly for several years after the patient has quitted the occupation which exposed him unduly, though the pre-rodent keratoses will have been manifest for a considerable period.

There is the family W. of eight surviving brothers and sisters, brought up on the Monaro, a district which produces a large number of rodent tumours and is high and cold, but bright and clear. Six of this family were treated by me for rodent disease or pre-rodent keratoses. They were all very fair


W
J. ♀, aged 60, Monaro, fair, rodent and keratoses, treated.
S. ♀, aged 56, darker skin, Monaro; no lesions.
T. ♀, dead, fair, keratoses.
J. ♂, aged 38, fair, farmer, Monaro, keratoses, treated.
E. ♀, died in infancy.
W. ♂, aged 64, farmer Monaro, very fair, large rodent, numerous keratoses, treated.
M. ♀, aged 58, Monaro, fair, keratoses, treated.
A. ♀, aged 54, Monaro, fair, keratoses, treated.
S. ♀, aged 40, fair, has lived in Sydney for 20 years. Shows no lesions.
R. ♂, aged 36, farmer, very fair, Monaro, keratoses, treated.

with one exception, all were born and all but one had lived all their lives on the Monaro. They are of mixed Irish and English blood. The family tree and description was given me by the eldest sister, one of the sufferers.

Thus all the three brothers had developed rodent or prerodent lesions; all were farmers; all were fair and the only surviving sisters who were unaffected, were one who had left the country for the city at twenty years of age and a second who was of distinctly darker complexion.

The brothers who were naturally more exposed, developed lesions earlier and it surely cannot be regarded as a mere coincidence that the dark sister and the sister who married a city man at twenty and had lived the last twenty years in Sydney, are the only members of the family to escape.

Clinical Observations on the Possible Transformation of Rodent Tumour into Squamous Epithelioma.

While not being able to relate an indubitable example of the transformation of rodent tumour into squamous epithelioma, the history of a slow growing, apparently rodent tumour, having changed into the more rapidly growing lesion which was present at the time of examination and which could be recognized as squamous epithelioma on both clinical and histopathological evidence, has come within my experience, rarely it is true, but still on enough occasions to make me strongly suspect that such a metamorphosis may take place.

PART II.

Histopathological Observations.

The Occurrence of Squamous Epithelioma in the Skin.

Localization.—Squamous epithelioma was found in specimens taken from almost all situations on the skin, but much more commonly on exposed areas, face, neck, hands and forearms. But it was remarkable that all lesions without exception, whether fully developed or in a preliminary stage, which were derived from hands or forearms, proved in microscopical characters to be of squamous and never of basal-celled type. This was true even when coincident growths taken from the face proved on histopathological examination to be rodent tumours.

When the specimen examined came from the face, a lesion of the coarse, prominent, cutaneous horn type always disclosed changes indicative of squamous epithelioma nature.

The histopathological characters of squamous epithelioma are too distinctive and too well known to need repetition, but examples of early squamous epithelioma are to be seen in photomicrographs Nos. 31, 32 (not reproduced).

The Origin, Development and Methods of Spread of Rodent Tumours.

The early downgrowths of rodent tumours such as are commonly found in sections of snippings of the flat finer type of keratosis mentioned above, are much more delicate than is the case with the digitations of beginning squamous epithelioma. This character is well shown in R45527/A (see Figure I.) which also displays the well marked hyperkeratosis and the proximity of these early downgrowths to a sebaceous follicle.

The later development may be deeper penetration and the formation of early clubs as is seen in the earliest definite rodent tumours of the series, R50540 (see Figure II.), R48980 (see Figure III.) and R48806 (see Figure IV.).

Another line of development seems to be that neighbouring digitations may fuse together or more likely there occurs a spread of the stimulus to neighbouring cells, so that curtain or festoon like structures are formed. The earliest example of this is to be seen in R47804/A/2 (see Figure V.) and a slightly later development in R45527/C (see Figure VI.).

Special attention is to be drawn in connexion with the above early lesions to several matters of importance.

(i.) In the first place there is the curiously frequent association of the early rodent lesion with a nearby sebaceous unit. This is seen in Figure I., in Figure II., in Figure V. and in Figure VI. This relation is to be noted again and again through the series and can be seen in another form and at a later stage in the curiously intimate and friendly fusion between a rodent mass (with a surface epithelial connexion) and the deeper parts of a sebaceous follicle (see Figure VII.).

(ii.) The second part is the much disputed hyperkeratosis which is shown in Figure I.; it was torn off in cutting (see Figure II. in R50540); it is seen about to be shed, owing to pressure from the underlying tumour in Figure III. and is largely shed in Figure IV. It is displayed well in Figure V. and again in Figure VI. It is elsewhere frequently noted, but perhaps nowhere better than in R50632, a small rodent tumour obviously arising from the epithelium and still covered by its keratotic cap. This specimen, however, was not photographed.

(iii.) Even in more mature tumours the epithelial connexion can be traced especially in those of the superficial cicatrizing type. The connexion with the surface epithelium of quite large club-like masses can be seen in R45505/12a (see Figure VIII.) and in R45505/4/1 (see Figure IX.). High power magnifications (× 250 diameters) of the stalk of the former section (see Figure VIIIA) and of that of R50540 (see Figure XI.) show the transition from basal cells of the surface epithelium by indistinguishable degrees into the definite tumour cells of the rodent mass.

The surface epithelium in the first two of these sections shows the atrophic change brought about probably by pressure applied from beneath by the underlying developed tumour.

Epithelial connexion of definite rodent masses can also be seen in R45506 (see Figure X.), in R48980 (see Figure III.), in R48806/1 (see Figure IV.) and in many other sections not photographed (for example in Berne No. 1, SU5712, R47775, R48691, M57 *et cetera*).

(iv.) The general histopathological characters of the rodent tumours comprised in this series do not afford any fresh information. The formation of clubs and their relation to the normal tissues they are invading, is and has always been well recognized. The placing on end or palisading of the outermost cells is quite evident in nearly all the rodent tumours of this series. It was hoped that this feature would serve to distinguish rodent tumours from the type of growth next to be described, but while palisading is certainly better defined and much more general in the former, still it was a very imperfectly developed feature in a few rodent growths and was distinguishable, though only in places and to a minor degree, in some portions of sections of the semicystic tumour.

So this arrangement of the outermost cells can be used only in conjunction with other variable characteristics as a means of forming an opinion upon the distinctive nature of the specimen to be examined.

One of the common characteristics of rodent tumour is that of formation of cavities. Abundant examples of this are present in the specimens of the series, though curiously enough only in R45505/12/a (see Figure VIII.) does the typical cavity come within any area photographed.

This cavity formation generally suggests origin by a kind of necrotic process resulting from the cutting off of food supplies in the central parts of rodent masses and the space is generally occupied by cell *débris*. But some specimens, as for example R47504, which is almost certainly of epithelial origin and exhibits such epithelial characters as whorling (see Figures XII. and XIII.), display the existence of smaller, irregular cavities which seem to some extent to foreshadow the type of cavity to be described later in connexion with the semicystic (? endothelial) tumour, SU2722 (see Figure XIV.).

Therefore the type of cavity formation, the size and shape and contents of cavities cannot be relied upon as distinctive of rodent disease as apart from other growths simulating it in clinical and histological characters.

Some notable variations are seen in the cell characters and behaviour of rodent tumours of this series and these serve at times as a help in differentiating rodent from other growths. For example, it is not so infrequent to find whorling of cells in a definite rodent mass and even imperfect cell nest formation. Examples of the latter are to be seen in R47504 (see Figure XIII.) and in R47775 (not photographed). Other examples of an attempt to form cell nests occur in the series and an arrangement of cells in whorls is still more frequent. Such an attempt at cell nest formation or even at whorling was not observed in the semicystic variety of tumour.

In some of the specimens, notably in R45506 (see Figure X.), where palisading was not a definite feature, the rodent cells tended to assume a more spindle form than is usual. The same characteristic (spindle formed cells) occurred in R48461, an epithelial tumour which has an air of greater malignancy than most rodent growths on account of the greater infiltrating tendency of the cell masses, though in the deeper parts of the tumour there is no sign of keratinization, even of a partial character such as is seen in the cells of a squamous epithelioma. The likeness to R45506 in respect to the distinct spindle form of the cells to some extent counteracts this impression, since the latter specimen is unmistakably rodent and of a low grade of malignancy.

The same spindle form of cell was met with in M64 and in SU2713.

This character is only an occasional variation and as the examples of it include at least one specimen of doubtful interpretation, no value can be attached to its occurrence.

(v.) One of the most striking observations was that of the obviously multiple points of origin of rodent tumours from the epithelium. As illustrations of this it is only necessary to point out that Figures VIII. and IX. are of two different sections from the serial cutting of the same rodent tumour (R45505). This was an occurrence frequently observed and examples of it at the spreading edge of a rodent tumour will be mentioned in the next sub-section.

The best example of multiple points of origin occurred in SU5712, in one section of which there were no less than nine such points of origin. To

observe this it is necessary to move the slide on the stage. This phenomenon, therefore, does not lend itself very well to illustration by photomicrograph.

(vi.) The next point of interest is the manner of spread of rodent tumour by involvement of the adjacent portions of epithelium in the fever of proliferation. The deeper penetration of the rodent tumour by fresh formation of buds from existing clubs and infiltration of neighbouring tissues thereby is the common method, but not the only one. When an examination is made of the edge of a rodent tumour and more especially of the edge of a rodent tumour of the superficial cicatrizing type, it is easily seen that the adjacent areas of epithelium are successively involved in the activity and proliferation. This results in downgrowths to form fresh rodent clubs distinct from the original tumour mass. It is almost as if there occurred an infection of the nearby epithelium.

In the superficial cicatrizing type of rodent tumour the penetration into the deeper parts, subcutaneous and deep fascial, seems to be held up for a considerable period and the original tumour cell masses die out leaving a cicatricial area, with a surviving island of rodent cells here and there, while the process of cellular proliferation and tumour production of the original rodent type continues as briskly as ever in the epithelium at the edge of the lesion. A definite illustration of this process is to be seen in photomicrographs 1, 2, 3 and Figure I. of R45506 which is a specimen taken from the edge of a rodent tumour of superficial cicatrizing type. These obviously new downgrowths from the skin epithelium covering the tissues in the neighbourhood of the original growth have no cellular connexion with the main tumour mass.

As mentioned in Part I., there occur occasionally examples of a skin tumour with clinical characteristics which serve to differentiate them fairly clearly from rodent tumours. Of this SU2722 certainly and R47505 and R48260 possibly are instances. As will be seen by reference to Figure XIV. the histopathological appearance of these tumours is one which in places (middle of reproduction) shows distinct similarity to some rodent formations, but which for the most part lacks the band of more deeply staining palisaded cells like an embroidered edge to a piece of needlework so generally characteristic of rodent tumour. Moreover, there is a great tendency to form cavities which look like the result of a distinct effort to produce tubes or acini and which are generally distinguishable from the cavity formation (by a process very like necrosis) that occurs in rodent club masses (see Figure VIII.). In some rodent tumours, however, there is a distinct suggestion of a finer cavity formation, as in R47504 (see Figures XII and XIII.) and in R48806 (see the top left hand corner of Figure IV.) which to some degree approximates to the more lace-like reticulate formation seen in Figure XIV., yet has an epithelial connexion.

But in SU2722 a more or less definite stroma exists, as shown in Figure XIV., with a division into lobules by the stroma, as distinct from the presence of rodent clubs pushing in amongst normal tissues.

No epithelial connexion can be traced, but this is, of course, a purely negative finding that does not exclude the existence of such an origin which might have been lost in the course of the extremely long, slow growth of the tumour.

The existence of a stroma, the rare and imperfect palisading of the outermost cells and the formed as distinct from necrotic character of the cavities, in addition to the clinical characters taken together seem to warrant the differentiation of this growth from rodent tumour.

Further reference to the interpretation of this tumour will be deferred to the later pages of the paper.

In addition to the tumour mentioned in the preceding paragraphs there also appears three times in this series a formation which for convenience I have called the "sebaceous" tumour and which is illustrated in R47504, R47804 and SU5648. In R47504 (see Figure XV.) the cells of this tumour can be seen arranged round and more or less filling the interstices between numerous sebaceous gland elements, though the tumour cells cannot be traced to an origin from these glands. The cells composing the growth are looser than rodent cells and show quite distinctive staining characters from those of a rodent tumour. They seem to be arranged in a somewhat lobular form round the sebaceous glands and do not form clubs like rodent cells do. This association and grouping and to some extent the cell characters suggest a possible origin from the cells of sebaceous glands.

Formation of similar nature but with a curious admixture of some large multinucleated cells, occurs in R47804 (see Figures XVI, XVII and XVIII) and in photomicrographs 35 and 38, this time in close proximity to a pilosebaceous follicle, but not with the same arrangement round the bodies of sebaceous glands as in the preceding specimen. What the significance of the multinucleated cells may be cannot be settled, except that they are not giant cells of inflammatory origin. Several of these can be seen in Figure XVII., as also can the tendency to lobulation which, however, is much better observed in photomicrograph 38 and Figure XVIII. The clear outlines of the cells and the distinct interspaces, as well as the staining properties, serve to distinguish these masses from ordinary rodent growths. And yet in both these cases in other portions of the same specimen are to be found typical and unmistakable basal celled epithelioma (rodent). By way of illustration it is only necessary to contrast R47504 (Figures XII. and XIII) with R47504 (Figure XV.) and R47804/B/1 (Figure XIX.) and R47804/A2 (Figure V.) with R47804/1/A1 (Figure XVI., XVII. and XVIII.) and this association is manifest.

Again in Professor Welsh's specimen SU5648 exactly the same type of cell is seen, the same tendency to lobulation, but this time there is no association with the sebaceous elements, the cells occurring just under a thickened surface epithelium.

ILLUSTRATIONS TO THE ARTICLE BY Dr. E. H. MOLESWORTH.

Figure I.

Figure II.

Figure III.

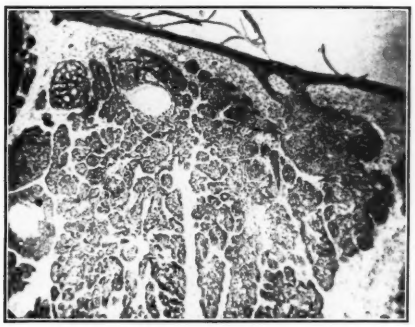


Figure IV.

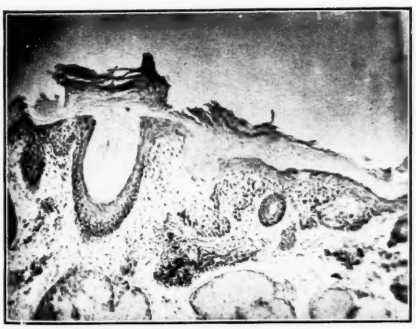


Figure V.

Figure VI.

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Figure VII.

Figure VIII.

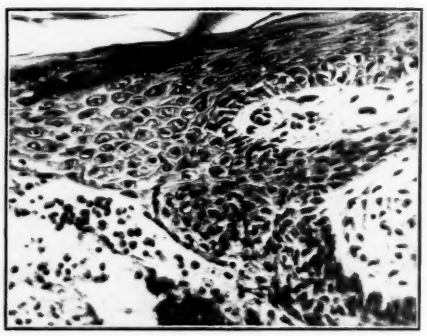

Figure VIIIa.

Figure IX.

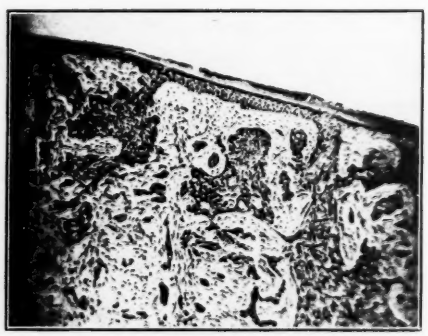

Figure X.

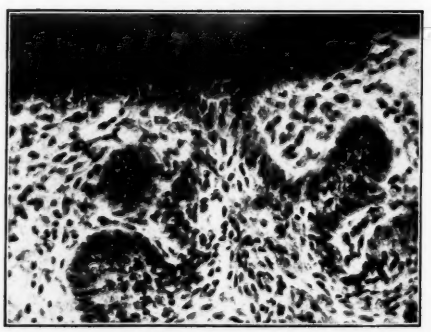

Figure XI.

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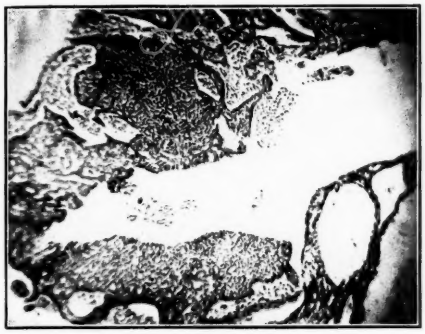

Figure XII.

Figure XIII.

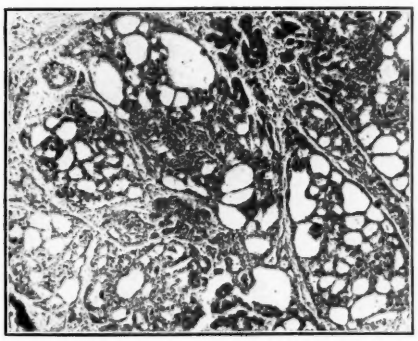

Figure XIV.

Figure XV.

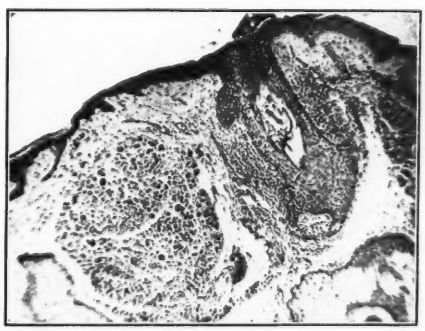

Figure XVI.

ILLUSTRATIONS TO THE ARTICLE BY Dr. E. H. MOLESWORTH.

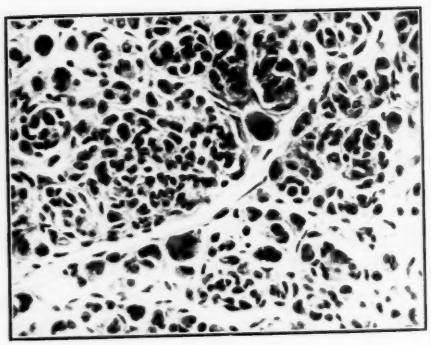

Figure XVII.

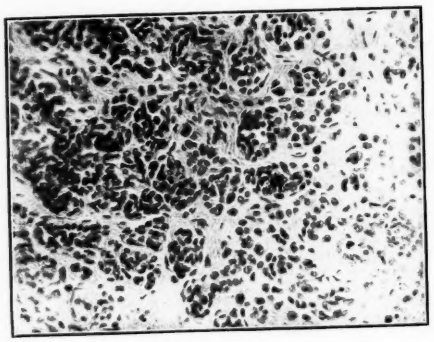

Figure XVIII.

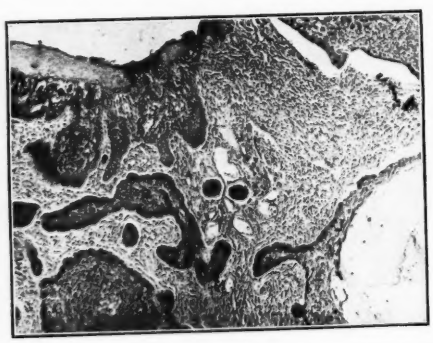

Figure XIX.

Figure XX.

Figure XXI.

In this specimen again no point of origin is seen and on this occasion there are no accompanying rodent masses.

Since writing the above the likeness of this formation to one form of unpigmented mole has been pointed out to me. There is a possibility of this being the explanation.

Observation on Mixed Tumours.

In addition to the occurrence of the cellular structure designated sebaceous tumour, it is necessary to describe a tumour belonging to the Pathological Department, University of Sydney. Sections of this (SU20/7/1908) were served out to students of one year as examples of endothelioma. Sections of some of the tissue remaining in the next year were again distributed, but on being stained a number of these were found to contain in addition to the tumour observed the year before masses of squamous epithelioma with typical cell nests showing an origin from surface epithelium. Reproductions of this tumour are seen in Figures XX. and XXI. (photomicrograph 26). The first shows the deeper staining cells of an underlying tumour which contains numerous small cavities, making contact with but apparently not originating from a hypertrophic epithelium. In Figure XXI. there is unmistakable epithelial downgrowth with cell nests, but only a few of the elements of the former tumour, which may be seen at the extreme right edge of the reproduction. The third microphotograph, 26 (not reproduced) contains incontestable squamous downgrowth occupying the whole field.

Whatever may be the true nature of the first tumour, endothelioma or rodent tumour, it is not possible to deny that it is entirely distinct in cellular character, staining, arrangement and manner of infiltration from the squamous epithelioma later cut into.

Although different pathologists have given different interpretations of the first tumour, they have all agreed that the abrupt change in cellular character and staining reaction of the fine pointers of the underlying tumour from those of the cells of the epithelium with which they come into contact, makes it almost impossible to believe that these points of contact are points of origin. The imperceptible gradations from typically normal cell elements of the epithelium into typical rodent cells seen in Figure VIII. (high power reproduction Figure VIIIa.) and in Figure II. (high power reproduction Figure XI.) do not exist in Figure XX. in which, on the contrary, an abrupt change is obvious.

A suggestion of the same sort of happening occurs in SU2956 (not photographed). This is a tumour excised from a child of three years of age. The deeper cell masses reveal likenesses to those in Figure XX., while at the edge of the ulcer a stream of apparently epithelial cells is observed (see full description of sections in appendix).

Apart from the histopathological appearance the extreme youth of the patient seems to exclude rodent formation as an interpretation of the main part of this growth (SU2956).

No incontestable example of mixed rodent tumour and squamous epithelioma comes into the series, but as the specimens were for the most part selected in an early stage of development, this is not surprising and too much weight must not be given to the evidence afforded by this series on this point.

PART III.

Statement of Physical Facts and Observation of Physical Effects on the Skin.

Attention has been drawn in an earlier part of this paper to the likeness between the changes induced in the skin which has received an over-dose of X rays or radium, namely hypertrophy, atrophy, pigmentation and teleangiectases and those appearing in the skin of some patients suffering from rodent diseaes who have never been exposed to such irradiation.

We know that such a combination, such a syndrome in fact, is due to radiation effect and that physicists agree that X rays and radium rays produce their effects by means of ionization of the substances irradiated.

It is an established fact that X rays and radium rays can provoke the production of squamous epithelioma. This event is familiar to all as a possible aftermath of a prolonged exposure to small doses of X ray. An account of the experimental production not only of squamous epithelioma, but also of sarcoma by imbedding a small quantity of radium in the tissues of mice is given in *The Lancet* of September 25, 1926, by Professor Frans Daels and Dr. Bacten, of the Belgian Cancer Research Council.

The conclusion that this tumour production is an effect of ionization follows as a necessary and natural inference and shows what importance must be attached to ionizing influences operating for a long time upon tissues, as provocative of tumour formation.

We know that sunlight unfiltered by cloud is rich in ultra-violet rays and it is a scientifically established fact that ultra-violet rays have a definite ionizing capacity, much inferior to yet comparable with the ionizing influence of X rays. We also know that the hours of sunshine are much longer and the sky much less clouded, especially in the inland parts of Australia, than is the case in any part of the British Isles.

The effect of ionization in the direction of stimulating the production and cellular proliferation of new growth has to be considered alongside the opposite effect of this agent, equally well established, namely a depressant action on cellular activity and destructive effect on the cells themselves when applied in large doses. This phenomenon is illustrated by the curative action of radium on the keratoses and small epitheliomatous lesions that develope on an area affected by radio-dermatitis. It is paralleled by the conflicting results obtained by the administration of a drug, for example alcohol, according as to whether it is used in small or large doses. The stimulating effect of a small dose has its opposite in the depressant and even fatal result of

a large dose. As with the drug, so it is with ionization which causes cellular irritation and stimulation in frequently repeated small doses, depression of cell activity, selective cell destruction or massive necrosis with varying grades of large dose.

Both the chemical agency of a drug and the physical agency of ionization can be relied upon to produce a sting like that of a flick from a whip, the stunning effect of a club or the destruction such as results from a blow with a sledge-hammer, according to the dose employed.

Another established and well recognized effect of ultra-violet rays is their curative action when employed for the treatment of *lupus vulgaris*. Finsen first employed simple concentrated sunlight for this purpose, but later modifications of the method have brought about the use of electric light from arc lamps, quartz lamps and the like, useful by reason of the richness of this source of illumination in ultra-violet rays and by reason that it is always available, while the use of sunlight makes regular treatment difficult or impossible.

This curative effect is manifested even when the tuberculous process is well established. We also know that tubercle bacilli will not thrive or even survive long when exposed to sunlight or to ultra-violet radiation which has a strong bactericidal action.

An observation which may deserve a place in this part is that such diseases which seem to owe their origin to insufficient ultra-violet absorption, are very rare in Australia as compared with England and that the use of ultra-violet irradiation as a therapeutic measure is very inferior in its results to the employment of this agency in Europe. These facts are not to be wondered at when we consider the rich actinic component of our abundant Australian sunshine.

PART IV.

Conclusions Drawn from Facts Bearing upon the Causation of Rodent and Skin Epithelioma Generally.

Taking into consideration the facts, collected from other sources or obtained personally and related in the preceding chapters, it is possible to advance a hypothesis which will reasonably explain the divergence in the incidence of the two diseases, lupus and skin epitheliomatosis in the same race under different climatic conditions and to show that the rarity of the one and the frequency of the other are in reality due to the operation of the same cause.

Indeed it is necessary to formulate some such hypothesis because already expressed theories cannot be made to fit in with the facts recorded.

It is true that in the case of *lupus vulgaris* it is generally suggested that the better conditions of living, greater prosperity, better food and housing of the people in Australia have produced this result. Whilst, however, admitting that such influences do operate in a favourable manner, as is evidenced by the smaller incidence of tuberculosis generally in British races domiciled in Australia as compared with that in the inhabitants of the Old Country, one cannot allow it to have been responsible for the practical non-occurrence of lupus in Australia.

For it must be remembered that pulmonary tuberculosis, for instance, while admittedly less frequent than in England, is still regrettably common in Australia and that joint and gland infection cause a great deal of death and disability in the Commonwealth.

If lupus occurred in Australia in the same proportion to the total number of cases of tuberculosis as it holds in England, we should have an appreciable number of cases of lupus developing in the neighbourhood of Sydney each year, instead of which we find one only in sixteen years. Hence it would seem necessary to seek an influence to explain the extraordinary rarity of lupus as compared with other forms of tuberculosis in Australia.

It is known that even in Europe not only the more prosperous section of the community, but that portion of the population which is occupied out of doors in the daylight and sunshine, is comparatively rarely attacked by *lupus vulgaris*. Reference has already been made at the end of the last part to the curative influence of concentrated sunlight containing a proportion of ultra-violet rays. The greater opportunities for outdoor occupations and recreations which undoubtedly exist even for the poorer members of the community and in the cities and industrial centres of Australia and the consequent greater exposure to sunlight with its ultra-violet rays, must be regarded as of prime importance and influence in inhibiting and preventing the development into lupus of the many inoculations of the skin with tubercle bacilli, which must be admitted as inevitable in view of the frequency of pulmonary and other forms of the disease.

It was the realization of the importance of this factor in the prevention of the development of lupus that led to the formulation of a workable hypothesis to explain the incidence of rodent ulcer. For if it be admitted that the particular influence preventing the development of lupus is the inhibition exercised by the abundant sunlight of Australia, a suggestion occurs to one's mind that this influence may also be responsible for other effects and amongst these for the extraordinary prevalence of rodent ulcer in Australia.

As far as is known to me this correlation has not been referred to in any publication, a fact which is not remarkable, since in few countries there obtain conditions such as in Australia—a country of bright sunlight inhabited by a comparatively prosperous population of outdoor habits, of generally fair complexion, recently transplanted from a climate characterized by long winters and cloudy days.

The occupation of India by Britain does not afford a parallel case since the English there occupy administrative positions and perform for the most part indoor duties and even in the army special

arrangements are made to avoid sun effects by making early mornings and evenings the hours of outdoor work and exercises and by providing special headgear for Europeans. Even there the incidence of rodent disease in Anglo-Indians seems to be greater than in the same race living in England, on the evidence of individual observers questioned upon the point, although no special reference in the literature has come to my knowledge.

In Australia no subject race survives, the aboriginal being nomad and free, unwilling to work and rapidly dying out, especially in areas where he comes in contact with whites. Hence all outdoor occupations, labouring, farming and pastoral have to be performed by the whites in possession of the country, a fair race as compared with the inhabitants of European countries, such as Italy and Spain where climatic conditions are comparable to Australian.

This, too, occurs in a country the greater part of which is still in the temperate clime, but singularly cloudless for the greater part of the year, in which men wear clothing similar to that of their brothers in England and hats designed and made for use in that country, with little or no fear of heat stroke or sunstroke, except on rare occasions and under unusual circumstances, a country where work goes on continuously through the middle of the day, under the full blaze of the sun in a cloudless sky for the greater part of the year and where the midday siesta has never obtained nor is likely to obtain.

The Texan cowboys and even the Mexican, a mixture of olive skinned Spaniard with the darker skinned Indian wear sombreros, while our farmers, graziers and outdoor workers persist in wearing the ordinary comparatively narrow brimmed felt hat, as made and used by their brothers in Great Britain.

Moreover, the Australian loves the sunshine and glories in it and is very liable to regard any precaution against sunburn as womanish and ridiculous. He waxes scornful at the expense of those more especially susceptible to the acute effects of sunburn. But he does not realize the remote effects of chronic sunburn or else he would himself take precautions which at present he derides.

It is to be expected that such deliberate flouting of the requirements of climatic conditions should produce an effect and the object of this work is to attempt to demonstrate some of those effects, as suggested by a realization of the reason for the scarcity of skin tuberculosis.

There was no inducement, owing to the extreme scarcity of material, to pursue the latter subject into its pathological aspects, even if there could be expected anything of new interest in such a research. But with regard to rodent ulcer the same conditions do not hold; material is superabundant and it appears on histopathological investigation that what we consider a clinical entity may be made up of several pathological varieties.

Moreover, the effects of chronic irritation by dust, chemical or physical agencies other than chronic sunburn, the development of cell rests and so forth will not explain the great preponderance of fair complexions among sufferers from rodent ulcer nor the demonstrable influence of an outdoor occupation in the production of epithelioma, squamous or basal-celled. Heat, dust, friction, trauma generally, if operative to any great extent in the stimulation of such growths, would bring about a comparatively even incidence in the fair and dark, the white and the black, a state of affairs that obviously does not exist.

Even without this consideration the utilization of the abundant material appears a scientific duty and even if the conclusions arrived at in this paper do not meet with acceptance, they may stimulate someone with a more scientific mind and more leisure than is available to a man in active practice to use the opportunities available for research into a condition which causes a great deal of suffering and of death.

Impressed then with the idea that the only possible explanation of the absence of *lupus vulgaris* in Australia is afforded by the realization of the action of the ultraviolet component of sunlight and appreciating not only the influence of ionization established in the case of X rays in the production of pigmentation, atrophy, hypertrophy and telangiectases, a combination which also occurs in association with rodent disease (see Part III.), but also the fact that ultraviolet rays, too, can and do ionize, it was borne upon me that ionization by this agency might cause rodent disease. And it was found that this explanation fitted in with all the facts and observations detailed in the preceding parts.

In support of the hypothesis there is much and strong evidence.

1. The disease occurs with much greater frequency in men who by their occupations and predilection for outdoor exercise and hobbies are much more exposed to sunburn, than in women.

My figures agree with those of Macleod[5] who gives twelve men and eight women out of twenty successive patients, that is three to two, whereas mine are twenty-three to fourteen, that is a little more than three to two.

2. Such women as are affected by rodent disease are seen on reference to the consecutive series of cases detailed in Part I. to be with one exception of outdoor habit and to show signs of exposure to chronic sunburn.

Apart from this series two cases remain in my mind as appearing at the time to be exceptions to this rule. They are as follows:

Mrs. M., a lady of ample means and sedentary habits (by reason of partial invalidism) was affected by rodent disease. It was not for some time that the information was forthcoming that her habit for twenty years was to spend the greater part of the day sitting in the sunshine either on the verandah or in a bay window, according to weather changes and her state of health.

Another apparent exception was an out-patient who had a rodent ulcer over the inner end of her clavicle. But this patient admitted having been occupied for many years in laundry work performed in her own home, exposed to

daily sunburning while drying the clothes in the open and being accustomed for the sake of freedom and coolness to fold in the neck of her blouse so as to leave a deep V-shaped opening from the shoulder to mid-sternal region, though she wore a hat which protected her face.

Thus two apparent exceptions go to prove the rule.

Even when women do follow outdoor occupations or hobbies they are much more disposed and accustomed by reason of vanity or care to wear adequate sun protection in the shape of wide, shady hats or veils which according to the present hypothesis, would protect them to a large extent. Even from early childhood and in poorer circumstances they are trained to take care of their complexions and in this case vanity appears rewarded by comparative immunity to rodent ulcer.

The outdoor occupations and habits of both men and women affected is a most striking feature of the series of consecutive cases mentioned above and cannot be explained as a chance observation.

3. The localization of rodent ulcer to areas of skin exposed to sunburn such as the face, neck and upper part of the chest, also supports the hypothesis.

4. The usual accompaniment of rodent disease by acknowledged sunburn effects such as freckling, keratoses and so forth (as shown in the parts in which the clinical and histopathological aspects are discussed) all tell in favour of the theory.

5. The types of complexion characterizing such a large proportion of rodent sufferers afford strong evidence in support (see analysis of consecutive cases in Part I.).

Browning under the influence of exposure to bright sunlight must have a purpose and it is obviously a protective effort, while freckling would appear to be an abortive though desperate attempt to provide protection on the part of a skin which cannot supply enough pigment to protect the whole threatened surface. Now, brown eyed people who generally tan easily and thoroughly very rarely and Italians and darker skinned races practically never suffer from rodent ulcer, while those types incapable of even browning as detailed in Part I. and exemplified in the W. family tree are heavily affected.

Indeed the consideration of these cases and of the exceptions in the family tree leave no doubt in my mind of the correctness of the hypothesis.

6. Support is also afforded by the analogy of squamous epithelioma of the skin with is precedent keratoses. These last only differ in degree from the prerodent keratoses. Darier describes the presenile keratoses as common in people such as farmers and outdoor workers[1] and rodent ulcer as especially prone to develope in association with these keratoses.[2] It is generally admitted that the warty keratoses preceding squamous epithelioma on the hands and forearms are due to chronic sunburn or at least to exposure. Macleod[3] even mentions these lesions as especially common in Australia, but only in association with the possibility of their further progression to the stage of squamous epithelioma.

But these keratoses, presenile and pre-epitheliomatous, are the same in substance even if different in degree whether they proceed to squamous or basal celled epithelioma. Men practising in diseases of the skin in Australia see them so commonly that we are very frequently able by familiarity so bred, to prophesy by the heaped up or flat appearance they present or by the localization they affect, whether the base on section will show squamous or basal celled epithelioma.

In addition to this it is to be remembered that while *xeroderma pigmentosum* is no doubt characterized sometimes by the development of squamous epithelioma, Darier in discussing the tumours arising in the course of this disease says: *"les epitheliomes sont tantôt tubulés"* (Rodent), *"tantôt lobulés"* (squamous). I have, moreover, in my possession a section of *xeroderma pigmentosum* obtained years ago at Berne in the laboratory of Professor Jadassohn which is most distinctly basal celled. Darier and Jadassohn rank among the greatest of living authorities on skin pathology.

Xeroderma pigmentosum with its basal celled tumours is admitted by them and all others to be due to exposure to sunlight of a congenitally ultra-sensitive skin.

I have never seen nor heard of the occurrence in Australia of *xeroderma pigmentosum*, the predisposition to which seems to be familial, but such a patient exhibits what is only the extreme degree of susceptibility of which the red haired, freckled individual represents the next grade.

7. Another analogy can be quoted in the case of epithelioma of the lip. This disease by some curious mischance is not recognized generally as the effect of sunburn even in quarters where it may be accepted that squamous epithelioma of the skin is often due to this cause. This is illustrated by what I would suggest is a false conclusion published in the daily press in September, 1926, purporting to be a finding of the Medical Research Council in England. It was related, with reference to smoking as a probable cause of cancer of the lip, that it was found that those occupations such as farming, outdoor labouring and so on where smoking was permitted, produced proportionally many more cases of cancer of the lip than did such avocations as coal mining in which smoking was prohibited. It was significant that the occupations mentioned in which smoking was prohibited, were all such as afforded protection from sunburn and all those mentioned as permitting of smoking were such as exposed their followers to this influence and its effects.

It is to be suggested that a further inquiry might profitably be made in order to explain why those people who follow indoor occupations, though very frequently continual smokers, are very rarely indeed attacked by epithelioma of the lip.

The old clay pipe and the heat and roughness of the stem used to be blamed for this tumour, but nowadays when such pipes are almost unheard of, cancer of the lip still claims its customary toll and

smoking is still blamed for it in spite of the fact that the modern vulcanite mouthpiece is certainly neither hot nor rough.

Of course, such a large proportion of men following an outdoor occupation smoke that smoking is a convenient peg on which to hang the blame, but smoking does not explain why in 99% of cases of cancer in this region, the disease affects the lower and not the upper lip, the mucous membrane of which is protected by its situation from sunburn. Nor does it explain why outdoor smoking should be so much more dangerous than indoor smoking, that in quite an appreciable number of cases the patient is a non-smoker or smokes very little, though almost all have followed outdoor occupations or hobbies and reveal obvious sunburn effects.

Moreover, it will be remembered that cancer of the lip always originates on the exposed part of the mucous membrane and not on the inner portion which must be admitted to be more exposed to the heat and chemical irritation of tobacco smoke.

I have frequently gone into such cases which are also common in Australia and almost invariably have elicited the history of frequent and painful sunburning of the lower lip and we are all familiar with the pre-epitheliomatous keratosis of the lip so like the sunburn keratosis, as seen on the skin.

Without, of course, insisting that ionization from sunburn is the one and only cause of epithelioma of the lip, I should like to suggest in view of the objections mentioned above and the frequently conceded influence of sunburn in the case of squamous epithelioma of the skin, that attention should be directed to possible causes other than smoking to permit of a logical conclusion as to the causation of the great majority of these cases. In the rare cases when women suffer from cancer of the lip it is invariably found that they are outdoor sunburned folk.

8. A reference to Darier's book on "Dermatologie" (pages 641 and 642) where is found expressed the nearest approach to realization of the cause of skin epitheliomatosis (basal and squamous-celled), will show how near he came to the "light," only to turn his back on it at the last. He says:

La cause de la localisation à la face de l'immense majorité des epithéliomes de la peau, n'est pas connue; elle tient sans doute pour une bonne part à ce que cette region est le siège d'election de la degenerescence senile et de la leucoplasie, qu'elle est particulièrement exposée aux traumatismes, aux inoculations de germes infectieux, à l'influence des agents atmosphériques, et surtout de la lumière qui parait exercer une action favorisante.

Quant à la cause directe de l'epithéliome et des tumeurs cancereuses en general, on l'ignore.

Certain difficulties in the way of the acceptance of the theory have to be met.

It may be pointed out that the growth provoked in the skin by X rays is always squamous epithelioma, never rodent ulcer. In reply to this attention is to be drawn to the fact that very severe exposure of an unusually sensitive skin to ionization by sunlight also produces squamous epithelioma. And as X rays are admittedly a much more powerful ionizing agent than ultraviolet rays and are not impeded in their action by the natural protective mechanism of the skin (pigmentation), it is to be expected that their effect will equal the most severe exposure of the most sensitive skin to ultraviolet influence. It is a matter of the degree of stimulation; a severe ionization is liable to cause squamous, a moderate ionization basal-celled carcinoma. Note also that *xeroderma pigmentosum*, admitted to be due to sun effect, displays both squamous and basal-celled tumours.

One is frequently asked to explain how it is that ionization can cure the damage occasioned by the same agent. Why, if X rays cure by ionization (as is admitted), can it be reasonably held that the disease, so cured, was caused by ionization?

These objections are fully answered, I think, by consideration of the varying effects of increasing dosage described in Part III. The stimulating, depressant or destructive actions of ionization can be called into play by increasing the dose, just as the same effects can be induced by smaller or larger doses of a drug like alcohol.

If any further explanation be required it will be amply supplied by the findings of Dr. Moppett of the Cancer Research Committee of the University of Sydney, which show such diametrically opposite effects as hyperplasia and atrophy produced by equal doses of X rays of different wave lengths.[4]

Not a few objections are based upon odd cases of skin tumour (which have not been examined microscopically) occurring in a patient of comparatively dark skin, who has undergone very little exposure to sunburn. Some of these tumours are, I think, not rodent disease at all, but are examples of the semicystic tumour (? endothelioma) described in Part II. which will be further discussed in Part V. Others are squamous epitheliomata, only masquerading as rodent tumours for which they are mistaken on account of the slow growth and comparatively benign course which so often characterizes such growths arising in the skin as distinct from the mucous membrane. These epitheliomata may result from the cicatrices of burns (as instanced in kangri cancer) or may be due to other causes. Even if they are of true basal-celled type proven histologically they are very rare and it does not at all endanger the hypothesis to admit that other irritant actions may be capable of reproducing occasionally what ionization effects frequently.

It is occasionally objected that definite rodent ulcer has been found in a patient of dark complexion. Apart altogether from errors mentioned in the previous paragraph other errors have to be eliminated, since people are generally judged to be dark or fair according to the colour of their hair. This method of distinction as mentioned in Part I. is fallacious. Even if the patient is brown eyed, signs of freckling must be looked for and account must be taken of the degree and duration of exposure which may have been sufficient to overcome the defensive mechanism. In any case it is perfectly certain that rodent disease in olive skinned races

and individuals is so exceedingly rare that the occasional exception can with equanimity be ascribed to a cause which operates only rarely in comparison with ionization.

At this stage, however, even at the risk of appearing to labour the point, I wish to return to the consideration of a criterion by which might be determined what is a fair skin. Long after the main part of this paper was written, there appeared in my rooms as a patient suffering from a minor ailment a girl with eyes and hair "black as the raven's wing," but she was freckled. When comment was made on this, she at once complained that she could not play outdoor games or enjoy the surf without freckling and asked if anything could be done to prevent it? In reality she was fair, in the sense that her skin was white and incapable of producing an even coat of protective tan, but conventionally she was the darkest of brunettes. If her lot in life had been to marry a small farmer, to help in the harvesting and to perform the many other outdoor duties of a farmer's wife, she in all probability would develope rodent disease. Such people are exceptions, of course, but not so rare as might be imagined. And it is necessary to recognize the existence of this type in estimating the susceptibility to rodent disease. In other words there is no criterion of fairness of skin but the whiteness of it and its incapacity to brown evenly.

It is true that rodent ulcer sometimes occurs in usually shaded areas, as on the forehead, under the brows, in the retroauricular fold. On close inquiry it is found that many of these ulcers occur in people who have the habit of going bareheaded or who are long subjected to reflected light, as in the case of sailors who burn to the cap band; it therefore appears that as much evidence in support as evidence against the theory is obtained from these very cases and the rare exception carries little weight.

It may be objected that the extreme rarity of rodent ulcer on the hands and forearms, areas much exposed to sunburn and the frequent occurrence of squamous epithelioma on these areas, while it supports the theory as far as squamous epithelioma is concerned, should make us look for an entirely different cause for rodent ulcer. Such a contention entirely neglects the influence of terrain which may be fruitful or unproductive when sown with certain seed.

That there may be and probably are other minor factors which operate in conjunction with ionization in the production of rodent disease, can be readily admitted. But there is no necessity to imagine that such factors can operate without ionization as the main agent, unless such a supposition were compatible with the facts detailed in Part I.

Some idea of the nature of the factor or factors may be obtained by recalling that even on the face rodent disease is especially frequent on the more actively sebaceous areas and that in this respect the hands and forearms are much less active than the face. Reference to the part in which the histological characters are discussed, will yield evidence of the association of early rodent lesions with sebaceous elements.

In reply to those who maintain that heat, dust, trauma, the development from cell rests and the like are to be regarded as responsible for rodent production, it is necessary only to point out how lamentably such theories fail as explanations of the unequal incidence in the dark and fair, in England and Australia, in the miner and the farmer, in the stoker and the deckhand and all the observations of Part I.

General Consideration of the Hypothesis.

From the foregoing it will be seen how reasonably and well the theory of causation by ionization explains all these anomalies and reduces them to order.

While it is probable that such influences as mentioned above may act in a contributory or supplementary manner, it must be very rare that they act without the cooperation of ionization.

It seems reasonable, therefore, to press for acceptance of the hypothesis that in the great majority of cases rodent tumours and squamous epitheliomata of the skin and lip are due to the stimulant or irritant effect of ionization and that this ionization is supplied by the effect of the ultraviolet component of sunlight. And if this hypothesis prove ultimately to be true, it will have added to our knowledge of the causation of disease and by application of that knowledge to the prevention of common and dangerous affections of people like the British inhabiting countries like Australia.

Beside the incidence of rodent disease and skin epitheliomatosis generally such a theory makes it clear how the process of evolution has determined that jet black races predominate in the tropics, since they are able to avoid the dangerous effects of too much sunlight, while as one goes north successive grades of colour are met, brown, olive to fair until one finds the very white skin which enables its owner to absorb enough ultraviolet and light rays to enable him to continue to live.

The black skin of the negro child in the northern States of America will not permit of penetration of sufficient sunlight to support health unless extra supplies are made available by artificial means. On the other hand the unpigmented skin of the northern European races allows the penetration of an excessive dose of sunlight and one of the effects is the production by irritation of the skin tumours discussed in this paper.

Therefore the distribution of colour is no accident but due to selective influences operating under the law of evolution.

PART V.

Conclusions upon the Question of Origin, Characters, Conduct and Spread of Squamous Epithelioma and Rodent Tumours.

As nothing remains to add to our knowledge of the point of origin of squamous epithelioma, nothing need be said on this question.

But on the subject of conduct of a squamous epithelioma arising in the skin proper as distinct from the mucous membrane, there is much to be said.

A strong protest must be entered here against the conclusion that a squamous epithelioma arising in the skin is necessarily as malignant and dangerous as a similar growth arising from the mucous membrane. While these epitheliomata arising in the skin may and not infrequently do involve glands, this occurrence is almost always a very late development, so that the practice of removing regional glands because a small tumour arising in the skin has squamous epithelioma formation, is unnecessarily severe and maiming. It is quite true that at times there arise extremely malignant and rapidly growing epitheliomata of the skin which early involve glands, just as it is true that there occur sometimes examples of slow growing epitheliomata arising from the mucous membrane, which only involve glands very late, but both cases are exceptions; it is as unnecessary to extirpate the regional glands in the case of early epitheliomata arising from the skin as it is unsafe and unwise to fail to do so in the case of an epithelioma arising from the mucous membrane. This observation is the result of a large clinical experience of skin tumours of which the histopathological characters and subsequent history have very frequently been examined. Procedures based upon tumour structure, without reference to the tissue of origin of the growth and to clinical experience, are frequently ill found and in my opinion the practice of extirpation of regional glands in the case of early squamous epithelioma arising in the skin is a good example of this mistake.

But in the case of basal celled epithelioma there has been in the history of pathology of diseases of the skin a theory which has recurred from time to time, which supposes that rodent disease is not an epithelial but an endothelial tumour. This has been supported on the ground that the tumour displays no cell nests, that the cells do not undergo keratinization, that cavities are formed and that the histological features and behaviour of the growth are widely different from those of known epithelial tumours such as squamous epithelioma. It is easy to miss the epithelial origin in developed rodent tumours, this being lost either in the course of ulceration or because the sections examined do not include the rodent mass and the stalk with its epithelial connexion. Where connexion between tumour cells and epithelium is observed, it is explained that these are merely points of contact and not of origin and merely portend the early ulceration of the tumour at that point. These and some other arguments are used in support of the endothelial origin of rodent tumour. But it will be seen that they are almost all of the nature of negative evidence and no demonstration of the origin of the tumours from endothelium has been possible.

Against this theory the opposite one of epithelial origin continually regains acceptance on positive evidence, both clinical and pathological.

In the way of clinical evidence of the epithelial origin of rodent tumour there is: (i.) The observation of prerodent keratosis, indubitably the expression of epithelial cell activity (see Part I. and compare Darier's presenile keratoses); (ii.) the influence of sunlight (demonstrated in Part IV.) in determining the occurrence and site of the lesion. It is much more difficult to explain why this agent, having been filtered by the epidermis without doing harm, should still in the smaller quantities remaining be able to exert such an irritant effect upon the underlying endothelium as to provoke a growth which it failed to do when operating in stronger force upon the epithelium in passing through. That the epidermis is susceptible to irritation by light is shown by the accepted influence of sunlight in the production of squamous epithelioma.

But the really weighty and I think conclusive evidence comes, of course, from the histopathological side of the inquiry. The very definite and unmistakable points of origin examined under high power magnification allow the drawing of no line of demarcation between basal epithelial and tumour cells and are demonstrated again and again from the beginning downgrowths down to the developed rodent clubs described in Part 2; they are illustrated in many of the photomicrographs. So positive and plentiful is this evidence that it outweighs easily the negative evidence favouring the endothelial theory, even if the tendency to whorling and the production of immature cell nests in some of the rodent tumours (see Part II.) did not detract from the value of the objection that rodent tumours do not display the common epithelial characters. The palisading of the outermost layers of rodent clubs is also, of course, so like the arrangement of the basal layer of the epidermis, that this too must be counted as a feature favouring the theory of epithelial origin.

But the evidence is so strong, even without these supports, that the epithelial origin of rodent tumour appears to be established beyond further doubt by the many young and mature rodent specimens described in their histopathological aspects in Part II. and in the appendix and it appears superfluous to push the argument further.

In the same way the strong and plentiful positive evidence afforded for the existence of prerodent keratosis (Parts I and II.), for the multiplicity of points of epithelial origin of a rodent tumour (Part II.), makes it unnecessary to do more than state them as proven facts. The same can be said of the occurrence of multiple rodent tumours of which clinical evidence available in Australia is overwhelming (see Part I.) and of which there is equally abundant histopathological confirmation. So commonplace is this occurrence of multiple rodent tumour in Australia that practitioners in diseases of the skin in this country find it difficult to believe that at least some English authorities should be able to maintain that rodent tumour is always single.

The demonstration of a manner of spread of one form of rodent tumour at least (the superficial

cicatrizing type), by involvement of adjacent epithelium in the fever of proliferation, is apparently complete, the instances of it given in Part II. and observed in other cases being of a positive character which will hardly permit of refutation.

This manner of spread is very interesting, affording as it does a puzzle as to the mechanism by which it is brought about. It almost appears as if the fever of proliferation were contagious and is evidence against the theory that cancer is bred from a mad cell with an imperfect memory as to its destiny, produced occasionally among the multitude of cells constantly being born in the body. For with this manner of spread the main tumour mass is being reinforced continually by exactly similar cells produced not by reproduction from its own cells, but from those of neighbouring epithelium. So one would have to imagine spread of the madness by contagion rather than by heredity.

The interpretation of the semicystic tumour described in its clinical aspects in Part I. and in its histopathological features in Part II. and in the appendix is extremely difficult.

There appears to be sufficient clinical and pathological evidence to warrant the separation of this tumour, SU2722 (Figure XIV.), from what we understand as rodent tumour, but whether there is enough to justify its classification apart from tumours of epithelial origin is an entirely different matter.

It has been interpreted as an endothelioma, largely on the character of its cavities, but while admitting that these are different in type to that usually shown by such formations in rodent tumours, it is not possible to prove that they are not an attempt at the production of acini and that their existence might indicate an origin from sebaceous or even sweat gland elements.

As there is so much room for divergence of opinion in the interpretation of this tumour, I am content to suggest that the evidence available is sufficient only to distinguish it from rodent tumour, but to leave the question of its nature and origin for future determination. Perhaps the examination of some young tumour of this variety will afford the solution.

The long and frequent examinations of the sections and consideration of the clinical evidence have left me personally without any real leaning toward an interpretation of it either as an endothelioma or as a tumour arising from one of the glandular elements of the skin.

Macleod[6] mentions, as noted previously, a cystic variety of rodent tumour, but contrary to his custom he fails to give an account of the histopathological structure of this variety. He describes cystic degeneration as occurring in rodent masses with colloid content. And this undoubtedly happens and I think explains the occurrence of such irregular cavities as occur in R47504 (see Figures XII. and XIII.) and possibly also the cavity formation in R47505 and R48260, even though the cavity formation in these last two is much more elaborate than the apparently chance formations in the former specimen. But the cavities, so formed in appearance, the peculiar cell character and the stroma seem to render such an explanation far fetched for SU2722 (see Figure XIV.).

If this tumour be recognized as an example of the benign cystic epithelioma occurring singly and the latter be regarded as a tumour arising from the glandular appendages of the skin and attempting to form acini, such an interpretation would be acceptable. If it be regarded as a lymphangio-endothelioma (an interpretation which Macleod rejects for benign epithelioma[7]), it must also be admitted that the argument has great weight. But I feel sure that it is not a rodent tumour and I suspect that if Macleod excised and examined a specimen of the cystic variety of rodent ulcer which he describes in its clinical aspects, a tumour arising without any history and unaccompanied by any signs of sunburn, he would find the same structure and come to a parallel conclusion.

I now come to the consideration of the significance of the (?) sebaceous tumour described in Part II. The peculiar characters of this formation can be well seen only in the actual sections, though the photographic reproductions serve to distinguish them from ordinary skin tumours. The question is whether the cells of this tumour originate from the surface epithelium from sebaceous glands or from endothelium.

Taking the evidence for and against the origin from surface epithelium we have in R47804 and R47504 the coincident existence of rodent tumour of surface epithelial origin and in SU5648 we have these cells lying just under the surface epithelium, though no point of origin can be detected. The tendency to lobulation might be regarded as foreshadowing the formation of rodent clubs. Against this, however, are the distinctive cell characteristics with different staining capacity and very definite intercellular spaces quite foreign to rodent habit.

In favour of descent from epithelial cells of sebaceous gland and not surface origin is the definite grouping of the cells round the bodies of sebaceous glands in R47504 (see Figure XV.) and the nearer approach to sebaceous cells than to surface epithelium as far as concerns cell structure and appearance. There is considerable support for this in R47804 (see Figure XVI.), but this support is lacking in the sections of SU5648. The attempt at lobulation might be ascribed to an attempt to form gland-like structures due to cell memory; the fusion of cells of definite rodent nature and surface epithelial connexion with cells of the deeper part of a sebaceous gland, as seen in R45505, might be brought forward in support. Just as we have seen the stimulus to proliferation spread to nearby healthy epithelium on the surface in the superficial cicatrizing types of rodent tumour (*vide supra*), so it seems reasonable to imagine that the same stimulus might affect the closely related epithelial cells of the adjacent sebaceous glands. The pro-

liferation would, if it occurred, probably determine the product of a rather different type of tumour cell, which might account for the difficulty of bringing into line the two varieties of growth seen both in R47504 and in R47804. On the other hand SU5648 while not contradicting this, yields absolutely no support to the theory.

Finally the question should be considered whether this tumour may not be due to endothelial proliferation, induced either by the same cause which brought about the coincident epithelial growth or by the spread of the stimulus from existing epithelial growth to neighbouring endothelium. The lobulated appearance would then be explained by an attempt on the part of endothelial cells to form tubules and it must be remembered that early endothelial cell masses do approximate to this appearance.

Against the endothelial theory, however, is undoubtedly the frank epithelial nature of coexisting cell masses, as seen in R47504 and R47804, and it seems that more evidence is required before one can accept the endothelial origin.

Of course, the evidence of each theory of origin is slender and conclusions on the available evidence would be extremely precarious. But on the whole, in view of the ever recurring suggestion of sebaceous gland association with rodent formations, in view of the cell characteristics of this tumour and of the distinct association, in two out of three of the specimens of the cells, with sebaceous glands and of the evidence of fusion of surface epithelial growth with deep sebaceous cells in Figure VII., the balance of evidence would seem to indicate that the cells of this tumour are of sebaceous gland origin. Especially does it seem more likely that this growth is of sebaceous epithelial origin when one considers the greater probability of similar cells being infected with this fever of proliferation affecting the surface epithelium, than that totally dissimilar cells like those arising from endothelium should be stimulated in sympathy with cells of epithelial origin.

There is, however, no proof that such is the case nor is there any proof that the cells of this tumour do not arise from the surface epithelium and therefore the matter must remain undecided until perhaps a fortunate section discloses their origin.

As mentioned in Part II. there is a possibility that this structure may be of the nature of one variety of unpigmented mole. Of course the coincidence of such a mole having occurred within the portions of tissue excised for a coexistent rodent tumour in two separate instances, is difficult to accept, but such chances do occur and without absolutely tying myself down to this interpretation, I must confess that I am greatly impressed with the likeness shown to Dr. Keith Inglis's specimens at Sydney Hospital.

Next comes consideration of the capacity to form cell nests and of how far one is justified in imagining that this formation is the sole prerogative of squamous epithelioma. Seeing that rodent tumour is derived from basal epithelial cells whose normal evolution is toward keratinization, it is indeed remarkable that their descendants in a rodent tumour do not remember their destiny and produce keratinized structures like cell nests. Indeed, it is much more remarkable that they should not than that they should do so. And in the specimens examined in this series, one not infrequently comes across imperfectly developed cell nest formation, varying from the tendency to form whorls to the production of imperfect horny seeds. This, too, occurs in specimens whose tumour structure otherwise and elsewhere exhibits undoubted rodent characters (see Part II.).

Hence it might be suggested that cell nest production is not pathognomonic of squamous epithelioma and exclusive of rodent tumour, but that it is a tendency of tumours arising from the surface epithelium, but much commoner and much more evident in the former than the latter.

The question of transformation of rodent tumour into squamous epithelioma is doubtful, because in the cases in which a growth which was regarded as rodent tumour, proves to be squamous epithelioma, as demonstrated by its histopathological structure, we have no specimen of the growth in its earlier stages to convince us that its earlier clinical appearance was not a mask. That this may be so is rendered obvious when a long series of specimens are examined, a procedure which reveals the fact that what a good observer took to be rodent tumour on clinical evidence, proved occasionally on section to be squamous epithelioma and *vice versa*. But if this change from basal-celled to squamous epithelioma does not occur, it would really be very remarkable, when, as mentioned above, we consider the origin of the rodent tumour and the destiny of the cells of its origin. In fact as in considering cell nest formation, we must regard the rarity of the change as far more remarkable than the possibility of the change itself. If the formation of cell nests is to be regarded as characteristic of squamous epithelioma only, then in R47504, in R47804, in R47775 and other specimens we have examples of this transformation.

But personally I regard these specimens as rodent tumours which still display a little cell memory destining them to form keratin and do not consider that my series shows this phenomenon, that is unless specimen SU20/7/1908 (see Part II.) be interpreted as a rodent tumour and as displaying this transformation into squamous epithelioma. The interpretation of this specimen is, however, much disputed and different pathologists have given very varied decisions upon it, so that it would be unsafe to draw too positive a conclusion and too rigid and narrow an interpretation upon what is undoubtedly a mixed tumour.

Whether this specimen displays the occurrence of a further change in the characters and behaviour of its cellular components or whether the presence of one tumour by spread of the fever of proliferation mentioned previously has induced the forma-

tion of another tumour from the covering epithelium is a question which it would be of use to decide.

The constitution of one-half of the specimen entirely of tumour cells of one variety and the very rapid change to cells of a different kind in the second portion of the specimen rather leads me to an opinion that the second explanation (diffusion of the stimulus) is the right one. And this opinion in turn rather impresses me with the idea that as the stimulus is more likely to affect like than unlike cells, there is some evidence of doubtful value, it is true, on the side of the interpretation of the original tumour mass being of epithelial rather than of endothelial origin.

Having regard to the histopathological features of the original tumour component of this section and the foregoing consideration of the greater likelihood of spread of the stimulus affecting related rather than unrelated cells, I am compelled to regard as more weighty the evidence for the epithelial origin of this first tumour, which would thus be interpreted as a rodent tumour and the whole specimen as an example of mixed basal and squamous celled epithelioma. But obviously this is an opinion open to argument.

Unfortunately, therefore, this series does not afford any convincing example of the change from rodent tumour to squamous formation, which might however be found by examining a series of late rodent tumours in distinction to my series which were selected principally for their youth, in order to determine the origin and manner of the growth.

That such a change does occur is my conviction, forced upon me by clinical evidence and its failure to occur would surprise me much more than its occurrence.

PART VI.

Summary of Conclusions and their Application.

The conclusions that have been forced upon me by the clinical and pathological evidence afforded by this series are as follows:

1. Ionization by the ultraviolet component of sunlight is the predominant factor in the production of rodent tumour and squamous epithelioma, whether of the skin or of the mucous membrane of the lip. This is not to be taken to mean that other influences are regarded as incapable of producing such effects, but only that the operation of such influences is rare in comparison with that of sunburn.

2. Rodent tumour, like squamous epithelioma, arises from the surface epithelium and is preceded by a hypertrophic change in the epithelium affected, resulting in the formation of a prerodent keratosis which is finer and flatter than that preceding squamous epithelioma. Examples of this are numerous in the series which also contains a number of actual rodent tumours still capped by their early keratotic covering.

3. Contrary to the teaching of some English authorities, rodent tumour is most certainly multiple, at any rate in a country whose climate is sunny, a fact which is common knowledge and most easily demonstrated in Australia.

4. A rodent tumour may and frequently does arise from several and many points. These multiple points of origin are amply represented in the series.

5. The superficial cicatrizing type of rodent tumour spreads by stimulation of the neighbouring epithelium into activity identical with that which produced the original tumour mass, by infecting it, as it were, with the fever which drove the original area of epithelium to proliferate. This is not to be taken to mean that spread of rodent tumour by direct infiltration by the proliferating cells of the main tumour mass is not the general method of progression of the growth.

6. It is demonstrated that the formation of cell nests is not the prerogative of squamous epithelioma, but in not a few rodent tumours a distinct tendency to this development is shown, though to a degree much inferior to that shown by squamous epithelioma.

7. It is possible and indeed probable that rodent tumour may undergo a change and allow the development of squamous epithelioma, though proof of this is not forthcoming from the sections included in my series of young lesions.

8. Confused with rodent tumour is a tumour commoner in Australia than Brooke's tumour and trichoepithelioma and distinct from all three, a translucent, semicystic, very slow-growing tumour which is possibly an endothelioma, and which has led to much confusion in the past and should be definitely sorted out from rodent, if not from the epithelial group of tumours.

9. Another tumour possibly arising from sebaceous glands, possibly a variety of unpigmented mole and of a character distinguishable from rodent tumour with which it was associated in two of the three specimens and from the tumour mentioned in the preceding paragraph, also occurred three times in the series.

10. Squamous epithelioma arising in the skin is commoner than is generally supposed, much less rapidly growing in the beginning than the same tumour arising in the mucous membrane, much slower to involve glands, so that radical measures of extirpation of the regional glands are only necessary and advisable in the later, more active stages.

11. Rodent tumour has not appeared in my experience on the hands or forearms, even though a tumour in these situations has been coincident with and apparently due to the same cause as definite rodent tumour or tumours on the face, but epithelial tumours in these areas are always squamous epitheliomata.

12. A strong impression remains in my mind that the pilo-sebaceous follicle has some association with rodent tumour production.

Several lessons of rather considerable importance from the social point of view are to be learned from consideration of this series, if the influence of ionization by ultraviolet rays of sunlight be accepted, as seems reasonable, as the principal and commonest stimulus to basal or squamous cell proliferation and tumour formation in the skin.

The first of these lessons is that chronic over-exposure to bright and direct sunlight of a skin incapable of good even protective browning is dangerous to health and life. Therefore people who are unfortunate enough to have such a skin, should either follow an occupation which does not unduly expose them to the influence of sunburning, or they should adopt adequate measures for shading the face and neck, just as they would adopt adequate protection from cold.

It is useless and dangerous for a red haired man to attempt to accustom himself to sunburning, for he will never develope a capacity for browning any more than he will "by taking thought add a cubit to his stature." More than this, it is almost certain that, if he obstinately persists in this attempt, he will in his middle age develope rodent tumour or even squamous epithelioma, possibly to his complete undoing. Such a man if a farmer or outdoor worker must be taught to wear a much wider brimmed hat than is now his custom, something approaching the sombrero in fact, and generally to adopt all reasonable expedients to minimize his exposure to direct sunlight. It is possible to prophesy that such precautions if generally adopted would very noticeably diminish the incidence of rodent ulcer and squamous epithelioma both of the skin and of the mucous membrane of the lip and since all these conditions are of grave import, a calculable diminution of death and disability would result.

The next lesson, a corollary to the first, is that the craze for sunbaking and the recent vogue of artificial ultraviolet light baths, while possibly of some advantage, though not nearly to such a degree as in Europe, have grave disadvantages as well. Like all good things, such as food and drink, work and play, this abundant bright sunlight, an asset, a means to health, prosperity and happiness if used in moderation, can easily be overdone, so that the inevitable disadvantages which accompany all good things within human reach and conception, become intensified and exaggerated.

And some are always more susceptible to the disadvantages of some good things than are others. So it is with sunlight and its ultraviolet component and moderation and care should be taught in the use of this as well as others. One is frequently met with a surprised protest, on warning a patient with rodent ulcer to avoid unnecessary exposure, that he never dreamed that sunlight could harm anyone. But such is undoubtedly the case and sunbaking on the beaches has already begun to contribute its crop of rodent tumours. It may be that the installation of ultraviolet baths in private houses as recently described in the press, will also produce a quota, unless as is to be hoped it prove but a transitory enthusiasm and go out if only to make room for the next fashionable panacea.

In conclusion I wish to point out that though in this work I have only rarely mentioned authority, the reason for this is not that I neglected or despised authority, but that little or no useful purpose would be served by discussing the different views which have been expressed and that it seemed better to start *de novo* with the abundant clinical and pathological material available and to attempt to form an independent conclusion rather than to follow one or other of the accepted schools or theories. If my conclusions have clashed with the findings of any particular school, they have at least been reached by a prolonged search of rich material and by mature consideration of the information obtained therefrom, with as little interference by prejudice or preconception as is possible to my mind.

References.

(1) Darier: "*Précis de Dermatologie*," 1909, page 337.
(2) Darier: "*Précis de Dermatologie*," 1909, page 637.
(3) Macleod: "Diseases of the Skin," 1920, page 249.
(4) W. Moppett: "An Investigation of the Reaction of Animal Tissues Exposed to Monochromatic X Rays of Different Wave Lengths," THE MEDICAL JOURNAL OF AUSTRALIA, April 11, 1925, page 364.
(5) Macleod: "Diseases of the Skin," 1920, page 1146.
(6) Macleod: "Diseases of the Skin," 1920, page 1146.
(7) Macleod: "Diseases of the Skin," 1920, page 1119.

APPENDIX.

THIS portion is devoted to a more or less detailed description of slides selected as being most relevant to the inquiry into the causation and origin of rodent tumour.

The first specimens were fixed in a fluid containing one cubic centimetre of glacial acetic acid and ninety-nine cubic centimetres of 95% alcohol. This solution, however, was found to render the horny material too hard, so that in many instances it was torn off the specimen in the process of cutting.

The later specimens were fixed in nine cubic centimetres of "Formalin," seventy cubic centimetres of 95% alcohol, five cubic centimetres of glacial acetic acid and eighty-five cubic centimetres of water, with the result that the keratotic covering of the majority of the specimens was retained and appeared in the finished preparation.

The staining was done with hæmatoxylin and eosin for tumour cells and a few sections were stained by Pappenheim's method. The latter showed a liberal sprinkling of plasma cells among the ordinary, small, round cells constituting the inflammatory exudate at the edge of the tumour.

The sections are grouped for description as follows: (i.) Those recognized as squamous epithelioma (two only selected), (ii.) those manifesting pre-rodent changes and very early tumours of rodent character, (iii.) rodent tumours of epithelial origin and ordinary club formation, (iv.) rodent tumours of superficial cicatrizing type, (v.) the growths revealing cystic characters, (vi.) the sebaceous group.

Only a limited number of specimens are described in this appendix, the remainder being less instructive instances of one or other of the types described.

Squamous Epithelioma.

R45504 (Notebook No. 1, Box [1]).—D.M., *ætatis* forty-six, female. The tumour is a typical cutaneous horn with infiltrated base, removed from the eyelid. The patient freckled on arms and gives a history of sunburning easily.

Sections of the base of the cutaneous horn show the coarser downgrowth of epithelium associated with squamous epithelioma, which contrast so strongly with those of rodent ulcer (illustration, photomicrograph 31).

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[1] Box refers to the numbered slide boxes used to store the slides.

R45653 (Notebook No. 1, Box 2) shows deep coarse digitations with cell nest formation, indicating early squamous epithelioma formation (illustration, photomicrograph 32).

Pre-Rodent and Early Lesions.

R45527 (Notebook No. 1, Box 1).—K.P., *ætatis* thirty-nine, female. Two typical pre-rodent keratoses were present on the forehead. The patient had a rodent ulcer cured by X ray on another part of the face. No note of exposure or skin character was taken.

Large patch cut into two (*a*) and (*b*). Small patch (*c*).

R45527/A, No. 1 slide, also R45527/A/5 (illustrations, Figure I. and photomicrograph 17) show great hyperkeratosis and parakeratosis. In the centre of the hyperkeratotic area there is a dense small round cell inflammatory infiltration under the whole of the epithelium. The basal layer of this has here and there fine or feathery downgrowths.

In relation to some of the sebaceous glands in the section are two masses of cells which are much more deeply stained than the sebaceous cells proper and have palisaded peripheral cells such as are characteristic of rodent masses (Slide No. 1, section nearest the label).

This section shows the "festooning" commencing and suggests the sebaceous relation of rodent tumour.

R45527/B (Notebook No. 1, Box 1) shows hyperkeratosis in places torn off in cutting. There is seen the same hypertrophy of interpapillary processes as in (A), well marked round-celled inflammatory infiltration and downgrowths of the basal layer especially in the neighbourhood of a pilosebaceous opening (Slide No. 1, section nearest the label).

R45527/C (Notebook No. 1, Box 1) shows large club-like masses of basal epithelium, especially on either side of a sebaceous follicle. These masses of cells show distinct variation in staining reaction and a rather less compact structure than the cells of the epithelium proper. A further development of the festoon structure seems to be represented in this section.

Small round cell inflammatory infiltration under these downgrowths is marked as usual. Illustration of this section is seen in Figure VI. (see R45527/C/5 special box—specimen slide).

Cross reference to R45527/C: Compare R47804/A2, Figure V. for festoon development.

R44228 (Notebook No. 2, Box 2).—H.K., *ætatis* sixty-one, had a keratotic lesion which was given an erythema dose of X rays fourteen days previous to excision.

Microscopic appearance.—Hyperkeratosis with thickening of epithelium and tendency to festooning. The staining properties of cells were interfered with, probably the result of irradiation.

Compare R45527/C.

R45227 (Notebook No. 1, Box 1).—W.H., *ætatis* forty-seven, a male, shows typical pre-rodent keratosis (untreated) on his left temple. This was excised for examination.

The microscopic appearance is similar to that in R45527, showing early proliferation of basal cells, especially at the neck of a sebaceous follicle, with production of digitate projections surrounded by dense inflammatory reaction. (Interpretation: The probable beginning of rodent formation; supports R45527.)

Rodents Showing Epithelial Origin and Ordinary Club Formation.

R45505 (Laboratory Notebook No. 1, Slide Box 1).—E.G., female, *ætatis* forty-one, date October 6, 1917. The lesion first appeared twenty (?) years ago as a horny patch on nose. The patient has lived inland until quite recently, was much exposed to the sun, has a skin incapable of even browning and was freckled as a child.

Typical superficial cicatrizing rodent ulcer on the tip of the nose, with a pearly ring at the border.

A snipping was taken to include a portion of the pearly ring.

The following is an abbreviated account of notes in Laboratory Notebook No. 1.

Slide No. 1 shows a very distinct rodent mass with exceptionally well marked palisading of the outermost layer of cells. (Rough pen sketch in Notebook No. 1.)

Slide No. 3 shows long digitations projecting downwards, exactly similar in structure to the rodent masses in the neighbourhood.

Slide No. 4 shows very well the pedunculated appearance of the neck of a rodent mass and its attachment to or origin from the basal epithelium. It is impossible to determine any distinction between the tumour cells and the basal epithelium.

Slide No. 7 shows hyperkeratosis and a basal epithelial origin for the same tumour mass mentioned in the description of the earlier slides, in which the mass is covered by thinned epithelium. This Slide No. 7 shows continuity with a pilosebaceous unit. This is one of the specimens that suggest sebaceous association (see Slide No. 7, fourth section from top of slide, Figure VII.).

Later sections show every mass of cells connected somewhere with the surface epithelium, generally by a very narrow stalk.

This tumour of long duration is remarkable for its slow growth and for the fact that apparently as the cicatrizing rodent growth spreads peripherally, the cell masses shrivel and the overlying epithelium becomes atrophic, while the fresh epithelium at the active edge seems to be stimulated into tumour formation in its turn, with keratosis and downgrowth of epithelium. This tendency to the involvement and stimulation of the epithelium at the edge is seen in other sections, as in R45506, Figure X.

For epithelial downgrowths at edge see R48980, Box 4, Figure III.

Illustrations.—Photomicrographs of R45505 series slides; Figure IX. and photomicrograph 12 of No. 4, first section, × 80 and × 250 diameters; photomicrograph 13 of No. 4, second section × 250; Figures VIII. and VIII.*a* of No. 12*a* × 60 and × 250 diameters.

The laboratory notebook has a rough sketch of Slide No. 1, a pen and ink sketch of Slide No. 4 and a pen and ink sketch of Slide No. 12*a*.

R50540 (Notebook No. 6, Box 4—key section No. 2). Clinical data.—R.N., *ætatis* seventy-one, male, had no great exposure to the sun. There were three different growths. (i.) On the bridge of the nase, (ii.) on the *ala nasi*, (iii.) on the cheek.

All three were excised.

No. 1 shows definite rodent formation with some indication of epithelial origin, but this is not definite. There is little or no palisading to be seen.

No. 2, a smaller tumour, shows rodent formation and a mass of rodent cells arising from the basal epithelium (see Figures II. and XI.). It also shows apart from this other rodent masses. Definite palisading is to be seen. This gives the best and earliest instance of rodent derivation from the surface epithelium.

No. 3 shows definite rodent masses with club formation, but no signs of source of origin. Very definite palisading is to be observed.

Compare R47775, R48691, M57, R48980 (Figure III., photomicrograph 41), R45505 (Figures VIII., VIII.*a*, IX. and photomicrographs 12 and 13), SU5715, SU5712 (nine or ten points of origin), R50632 (still a keratotic cap).

M57 (Notebook No. 4, Box 4), from Dr. Nesbit's patient; a typical rodent with club-like masses and palisaded outer cells. It shows a definite point of contact and probable origin from epithelium with indistinguishable transition from normal to tumour cells. Some cavity formation is seen.

Epithelium covering the tumour mass is thinned possibly by tension.

Compare R50540 (Figures II. and XI.), R47775, R48691.

R48980 (Figure IV., Box 4, illustrations Figure III., photomicrograph 41). Clinical data.—C.B., *ætatis* forty-nine, a male, complexion fair and freckled, much exposed to the sun, shows a lesion of the neck.

Microscopic appearance.—Rodent masses with typical palisaded periphery, definitely attached to and apparently arising from the epithelial layer. The epidermis is very

definitely thickened and hyperkeratotic with necrosis just beginning in the centre.

Compare R50540 (Figures II. and XI.) and for similar tumours see: R47775 (Notebook No. 3, Box No. 4), R48691 (Notebook No. 3, Box No. 4), M57 (Notebook No. 4, Box 4), R45505/12*a* (Figures VIII. and VIII.*a*), R45505/4/1 (Figure IX., photomicrographs 12 and 13).

R47775 (Notebook No. 3, Box 4). Clinical data.—H.T., *ætatis* sixty-nine, a female, had a pearly ring of typical rodent appearance low down on her neck, with a nearby minute satellite patch (excised).

Microscopic appearance.—It is a beautiful little tumour bulging the epithelium outwards. The epithelium immediately above the growth is thinned, but evidence exists of keratosis just outside the tumour region, which was partly destroyed and torn off in cutting. There is definite continuity with and almost complete proof of origin from the epidermis in these sections.

Another interesting point is that though the deeper cell masses are very distinctly rodent in the type of their arrangement, of their staining capacity and of their tendency to palisading of the outermost layer, just under the epidermis there are several unmistakable whorling cell nests.

Compare R50540 (Figures II. and XI.), R48691 (Notebook No. 3, Box 4), M57 (Notebook No. 4, Box 4), all illustrating the epidermal origin of rodent tumour.

R48691 (Notebook No. 3, Box 4). Clinical data.—N.O., *ætatis* forty-eight, a male, of very fair complexion, very susceptible to sunburn, followed an outdoor occupation.

Microscopic appearance.—A very beautiful specimen showing definite rodent formation with excellent palisading of the outer layer of cells and fairly well marked cavity formation.

The epithelial origin is almost incontrovertible, connexion with a hyperplastic epithelium being obvious.

Compare R50540, R47775 (Notebook No. 3, Box 4), M57 (Notebook No. 4, Box 4), all illustrating the epidermal origin of rodent tumour.

In the deeper layers there is a distinct likeness in the formation of the masses to the tumours of the semicystic group. But palisading, though not very pronounced, is present even at ends of the large lobules furthest from the surface. The overlying epithelium is by now largely thinned and atrophic over main tumour mass probably by tension.

This is a specimen showing the close resemblance of a rodent tumour to the group of which SU2722 is the prototype (the difference consists in the palisading and connexion with the epithelium of the skin).

Contrast with SU2722 (Figure XIV.), R48260, R47505.

Berne 1 (Professor Jadassohn's specimen).—Tumour of definite epithelial origin forming large clubs and masses with some palisading, no cell nests and some similarity to R45527/C in cell character.

This specimen is also noteworthy on account of the demonstrably multiple points of origin.

Compare for multiple origin, SU5712, R45506 (Figure X.).

R50632 is a small rodent growth obviously arising from epithelium and still covered by its keratotic cap. A good specimen to convince those who doubt the existence of prerodent keratosis.

R49167 is an early rodent tumour showing epithelial origin and downgrowths multiple and interlaced. One slide in special box.

SU2140 appears to be a rodent tumour palisaded. No epithelial origin is to be seen.

R48806 (Notebook No. 4, Box 4). Clinical data.—J.M., *ætatis* forty-nine, a male, a farmer with fair complexion, constantly exposed to the sun; he has other rodent lesions. The specimen was excised from his forehead.

Microscopic appearance (see Figure IV.).—Rodent tumour, with numerous lobules, showing a point of contact and apparent origin from the epidermis. At the top left hand corner there is cavity formation as in R47805.

SU5712 is a beautiful example of epithelial origin of rodent tumour, with a remarkable number of points of origin (nine or ten) on the one slide. It forms the best example of this feature in the series.

Compare R50540 *et cetera* for epithelial origin (Figures II. and XI.). Compare with Berne No. 1, R45506 (multiple origin).

R45771 (Notebook No. 1, Box 2).—M.C., *ætatis* sixty, a male, has a typical rodent growth with a pearly ring. This lesion is crusted but not ulcerated.

The sections reveal well established typical rodent formation, but this is too advanced (with necrosis at the surface) to be of use to demonstrate the origin of the tumour.

R47655 *a* and *b*. Clinical data.—R.H., *ætatis* fifty-three, had a definite rodent growth under his right eye cured by exposure to X rays. The two lesions excised were small depressions, one on temple and the other on eyebrow, surrounded by a pearly ring.

Microscopic appearances.—In *a* there are definite rodent club-formed masses with palisaded cells at the periphery, but the lesion is already too far advanced to show the origin of any of the masses. This lesion shows some tendency to whorling of the cells in the club-like masses, an instance of the approximation to squamous epithelioma with cell nest formation. In *b* there are also definite rodent club masses, but there is no indication of the origin of the tumour. No whorling is seen in the sections of this specimen.

The existence of multiple rodent tumours is frequently denied by English authorities and this case though equalled and excelled by many others in the number of rodent lesions developed, is still a proof of the multiplicity of these neoplasms in one patient (three in all).

SU1090 (served out to class as endothelioma, compact structure). Very little palisading is to be noted, but otherwise there is little to distinguish it from other undoubtedly rodent epithelial tumours without obvious epithelial connexion.

Compare with accepted rodent tumours and contrast with SU2722 (Figure XIV.).

Rodents of Superficial Cicatrizing Type (Some with Spindle Formed Cells).

R45506 (Laboratory Notebook No. 1, Slide Box 1).—F.B., *ætatis* forty-five, a female. On October 6, 1917, a snipping including pearly ring left after insufficient surgical scraping and X ray treatment was examined. This patient was said to be dark eyed, but no history of exposure to sunburn was taken nor of the character of skin.

Slide No. 1 reveals epithelial activity not at one focus, but almost evenly along the surface of the section.

In several sections definite epithelial (rodent) formation is found; there has been no attempt at cell nest formation, but on the other hand no palisading of the outer layer of cells in each mass. The cells are rather spindle formed.

This section yields evidence of the epithelial origin; some of the divergence from ordinary rodent appearance may be due to insufficient X ray treatment. But owing to the above fact undue weight is not put upon this section.

Photomicrograph No. 1 of R45506, Slide No. 1, Section No. 3 × 60; photomicrograph No. 2 of R45506, Slide No. 1, Section No. 2 × 60 (a view of another portion of surface); photomicrograph No. 3 of R45506, Slide No. 1, Section No. 2 × 80. Figure X. of R45506, Slide No. 1, Section No. 3 × 80 (to show wide base of tumour growth along whole surface of the section).

Laboratory Notebook No. 1 contains a rough pencil and pen drawings.

Compare with R46939 epithelial tumour, R48461 rather spindle form, R45506 epithelial tumour (Laboratory Notebook No. 2, Box 3); Whitfield's "Escall" and SU2713 (Notebook No. 6, photomicrograph 15).

R46939 (Laboratory Notebook No. 2, Box 3). Clinical history.—A.C., *ætatis* thirty-eight, a female, had a typical superficial cicatrizing rodent tumour of three years' duration of the type seen in R45506, somewhat spindle form, palisading at the edge not distinct though present in parts. In Slides 1, 2 and 3 the streaming appearance is most evident, while in Slide 4 the ordinary rodent appearance begins to show, especially in thicker part of the mass and

in Slide 5 some club-like masses with well palisaded cells at periphery are to be seen.

It is these superficial cicatrizing lesions that tend to show spindle-like form of cells and few club-like masses.

Compare R45506 (spindle form of cells), Figure X.

M64 (Laboratory Notebook No. 5, Box 5).—This is a tumour of epithelial origin; there are no cell nests, no palisading nor club formation. It contains spindle-shaped cells.

Compare with R48461 (photomicrographs 5 and 6), R45506 (Figure X.), SU2713 (photomicrograph 15): spindle-shaped cells.

R48461 (Laboratory Notebook No. 3, Box 4, photomicrographs 5 and 6). Clinical data.—G.N., *ætatis* sixty-eight, a male, had a growth in the left temporal region. Other details are missing.

From the microscopic evidence it appears that there is a definite origin from the squamous epithelium which is stripped off for the greater part of the section. Strands of epithelial cells assuming rather a spindle form are seen to be infiltrating deeply.

The formation of the growth and appearance of the cells is reminiscent of R45506, but the infiltration is deeper and more threatening. There is no cell nest formation.

There is a question as to the reality of the apparent high degree of malignancy when considered along with comparatively benign R45506.

Compare R45506 (Figure X., photomicrographs 1, 2 and 3, Notebook No. 1, Box 1), M64 (Laboratory Notebook No. 5, Box 5), SU2713 (photomicrograph 15): spindle form of cells.

R45550 (Laboratory Notebook No. 1, Box 1).—A.S., *ætatis* forty; a snipping was taken of a rodent growth of eyebrow recurred after X ray treatment. It was originally of a superficial, cicatrizing type and reacted very slowly to radiation and the present recurrence appeared six months after cessation of treatment. Sections reveal infiltrating strands of cells of rodent type. As the specimen is a recurrent tumour no evidence of origin need be sought.

SU2713 (Laboratory Notebook No. 6, Box 5, photomicrograph 15), from a patient of dark complexion, is a section of a small epithelial tumour of rodent type obviously of epithelial origin. It is somewhat reminiscent of R45506 No. 1, second section, photomicrographs 2 and 3, as far as the shape of cells is concerned.

This is apparently an exception to the rule, but the particulars are meagre and the name of the patient suggests the possibility of the dark-haired but fair-skinned Irish type.

Compare R48461 (photomicrographs 5 and 6).

The Semicystic Group.

SU2722 (Laboratory Notebook No. 6, special box) is a specimen which came from a patient of dark complexion, unaccompanied by keratoses. This was a single lesion which had existed for fourteen years and only just at the time of excision had any threat of ulceration occurred.

The cells of the tumour are slightly different in appearance from those of rodent growth, without this difference being such as can be described in words, unless the expression "velvety" conveys the impression of the difference. But the elaborate system of cavities which have the appearance of having been formed rather than of having resulted from a necrotic process as in rodent tumour, is the most striking feature. It is this that has led to an interpretation of it as a lymphangio-endothelioma.

In the section photographed (Figure XIV.) there is a mass of obviously squamous epithelium away from the surface, but also apart from the growth. There is in this mass what appears to be a double cell nest, but as there is no inflammatory exudate in its neighbourhood, it is probable that this is a normal skin structure, probably a cross-section of a pilosebaceous follicle.

There is some palisading of the cells here and there, but again somewhat different to that of rodent tumour.

There is a definite fibrous stroma which is foreign to rodent habit and this together with the elaborate lacework of formed cavities and the velvety appearance of the cells supports the contention that this is not a true rodent growth. The clinical history and clinical signs of such tumours also lead to the same conclusion. But whether it is a lymphangio-endothelioma or a tumour arising from one of the glandular elements of the skin and endeavouring to produce acini, I confess I am unable to decide for myself and must leave the solution to others.

In Figure XIV. a typical field of this tumour is seen.

Compare R48260, R47505.

R47505 (Laboratory Notebook No. 3, Box 3). Clinical history.—L., a male, *ætatis* forty, sustained a small injury to cheek some years ago and on the site of injury there arose a small, slowly growing tumour.

Microscopic appearance.—It is an advanced tumour distending, but not touching the epithelium, which is normal in thickness and not keratotic. There is little or no inflammatory, small round-celled infiltration as seen in rodent tumours of epithelial origin. The tumour is composed of masses of cells staining like rodent cells, with innumerable cavities the walls of which are often only one cell thick. This characteristic is so marked in places that the tumour cells are arranged in a reticulate or lace-like manner. Moreover, the cells at the periphery of the masses are not arranged on end in the palisade manner characteristic of rodent tumour, as seen, for example, in R47504 and other sections with demonstrable epithelial origin.

It would seem that this specimen might be an example of the semicystic tumour which resembles rodent tumour in microscopic appearance, but it is not so distinctive as SU2722 (see Figure XIV.).

The stroma of this specimen is not so pronounced as that of SU2722 (Figure XIV.) and with the character of its cells so like that of a rodent growth may be regarded as a rodent tumour with cystic degeneration as described by Macleod.

Compare with SU2722, R48260.

R48260 (Laboratory Notebook No. 3, Box 4). Clinical data.—W. E., a male, *ætatis* forty-one, of dark complexion, has a lobulated, irregular tumour about the size of a large split pea on the left cheek, not ulcerated.

Microscopic appearance.—Large masses of tumour cells not unlike rodent tumour but not so well defined are present. There is no palisading, but a distinct tendency to cavity formation and reticulate or lace-like formation as seen in R47505 and SU2722.

This tumour may be another instance of the group of which SU2722 is the best example.

Compare SU2722, R47505.

R20/7/08 (Laboratory Notebook No. 6, Box 5, class block, Sydney University Pathological Laboratory), issued in 1919 as endothelioma opening out into cavities. Portion of the same block was issued in 1920; when after a few sections were cut, it was discovered that definite squamous epithelioma with cell nests was to be seen in the sections.

This is a most puzzling specimen as one has to admit the epithelial origin of the squamous epithelioma with cell nests and one begins to wonder if the so-called endothelioma tous formations were not also of epithelial origin. Against this conclusion is the fact that in such case the latter must be regarded as being of rodent nature and of epithelial origin and it must be admitted that squamous epithelioma occurred simultaneously with rodent tumour in the same area which is a coincidence hard to believe, or that further change occurred in the rodent mass leading to squamous formation or that a rodent growth by spread of the stimulus provoked the growth of squamous nature from neighbouring epithelium.

It is rather more difficult to imagine that squamous epithelioma might arise in an area already diseased and damaged by endothelioma, than that the same tumour might develope on an area diseased and damaged by rodent, a kindred growth.

But the histological evidence is not strong enough to enable the puzzle to be solved and unfortunately the clinical data available are of the scantiest and do not help.

Therefore it seems as if this puzzle must remain unsolved for the present, though it must be admitted that in the one block of tissue there exist two very distinct tumours, for whether the first detected growth be called rodent tumour or endothelioma, it must be admitted that the definite squamous epithelioma disclosed by later cutting is very foreign to either, though less so to rodent tumour than to endothelioma.

Illustrations of the two tumours occurring in this specimen are found in Figures XX. and XXI.

SU2956 (Professor Welsh's specimen) from a child of three years of age, shows infiltrating tumour cells below a depressed ulceration and, about the middle of the section cavity formation. There are also shown long epithelial downgrowths forming projections of epithelium into the tumour. At the edge of the ulceration there is seen a stream of cells of apparently epithelial nature gradually giving place to others of more endothelial appearance. This might be interpreted as showing the epithelial origin of the latter.

Professor Welsh interprets this streaming and the larger digitations as being due to secondary stimulation of epithelium by the underlying endothelioma.

The endotheliomatous masses in this growth, however, are very different in appearance from those seen in SU20/7/08 (Figure XX.) and still more so from SU2722 (Figure XIV.).

"Sebaceous" Group.

R47504 (Laboratory Notebook No. 3, Box 3). Clinical notes.—A warty growth of the face was removed by the casualty surgeon. No other history available.

Microscopic examination.—This is a tumour growth adopting different forms. For example on one side of a pilosebaceous opening there are definite rodent clubs with some cavity formation and palisaded arrangement of the outermost layer of cells (Figure XII.). On the opposite side of the pilosebaceous opening there are several immature cell nests (Figure XIII.).

Finally in a detached portion of the section there are multiple sebaceous gland elements each surrounded by a number of epithelial cells which have somewhat the same characters as the cells composing the sebaceous glands, but which appear to be infiltrating the surrounding tissues (Figure XV.).

This specimen is very interesting as showing an apparent bond of union between a rodent-like tumour with squamous epithelioma on the one hand and a sebaceous gland tumour on the other.

Compare R47804, R45527/C, SU5648, sebaceous group; SU20/7/08, No. 2, class specimen, squamous tendencies.

R47804 (Laboratory Notebook No. 3, Box 3). Clinical data.—E.C., a female, *ætatis* fifty-two, had two small lesions, one on cheek; the larger was a clinically definite rodent tumour; the second on the eyelid was only keratotic and a third (larger) on right side of the skin of the lower lip. The patient had been much exposed to sunburn.

The larger lesion was cut into two, a^1 and a^2. The two smaller *b* and *c* were not divided.

Microscopic appearances.—In a^1 a mass of infiltrating cells is seen surrounding a sebaceous gland. The mass is loose and divided into strands or lobules, the latter probably cross sections of strands. The epithelium over this mass is thinned apparently by tension, but at the edge there is some hyperkeratosis. There is no continuity between the infiltrating cells and the epithelium in this section.

The structure of the mass and the characters of the individual cells are the same as those of the detached portion in R47504 and as the specimen is only a portion of the lesion of which a^2 is the remainder (see below), the parallel is almost exact.

Figures XIV. and XVII. and photomicrographs 35 and 38 and Figure XVIII. of R47804 a^1 specimen slide show the appearance of these cell masses. In Figures XVI. and XVII. some multinucleated cells are seen, the only occasion where such a phenomenon was observed in growths of this kind.

Compare with R47504 (detached portion), R45527/C and SU5648: sebaceous type for later discussion.

R47804 a^2 (Laboratory Notebook No. 3, Box 3) is a tumour showing definite hyperkeratosis and underlying this on either side of a pilosebaceous orifice hypertrophic changes in the epithelium, making a curtain-like formation or festoon of cells growing downwards (Figure V.). This downgrowth is surrounded by dense round celled infiltration. The cells of the downgrowths are rather paler and more spaced than are the ordinary epithelial cells, but are obviously of epithelial origin.

At the extreme end of the section is seen a patch where epithelial digitations are growing down (photomicrographs 28, 29, 30). One of these digitations is seen to be subdivided into three, foreshadowing club formation like rodent growths and another neighbouring digitation which has an imperfect cell nest.

Therefore in this specimen, as in R47504, we get the remarkable association of rodent clubs, incipient cell nests and a suggestion of squamous epithelioma, together with the peculiar tumour with relation to sebaceous glands, consisting of cells of rather sebaceous type.

R47804 a^2 is one-half of a snipping of which a^1 is the fellow.

R47804/B (Laboratory Notebook No. 3, Box 3) is an epithelial growth consisting of dense masses of cells arranged in club formation with cells at the periphery palisaded. Over the main mass the epithelium is beginning to necrose, but a very probable origin from the surface epithelium is shown (Figure XIX.).

In No. 3 serial slide—all sections—is seen a festoon arrangement of epithelium just outside the area of necrosis and connexion between the largest mass of typical rodent cells and the festoon.

In No. 4 serial slide full connexion of this club-like mass with the festoon can be discerned and also other digitate projections of epithelium very long and obviously part of the tumour, which are connected with other clubbed cell masses of the tumour.

These connexions run through No. 5 serial slide in which there is also a mass of clear spaced cells reminiscent of those in sebaceous tumour in R47804 a^1.

R47804/C (Laboratory Notebook No. 3, Box 3) is a small tumour of compact character, of definite epithelial origin yet with a strong tendency to the formation of club-like masses and to palisading of the peripheral cells (photomicrograph 34). The whole tumour appears to be a development of the festoon or curtain like formation mentioned elsewhere and as seen in miniature in R47804 a^2 (Figure V.) and in a slightly more developed form in R45527/C (Figure VI.).

Consideration of histopathological findings of the different specimens included in R47804 a^1, a^2, *b* and *c*.

This case seems of great importance because in three different lesions excised at the same time from the face of the same patient and of identical clinical appearance except as to size, there appear three different types of tumour, the typical rodent in *b*, in a^1 the peculiar sebaceous-like cells and in a^2 early epithelial downgrowths sometimes approximating the sebaceous type, sometimes to the rodent type and sometimes to the squamous type, while in *c* there is the fuller development of the festoon proliferation seen in other early cases.

No. R47804, therefore, seems a key to the different types of rodent growth, excluding, of course, the cystic tumours and also provides evidence of the epithelial origin and association with sebaceous elements.

The epithelial origin is shown better perhaps in R45505 (Figures VIII., VIII.*a* and IX., photomicrographs 12 and 13) and in R48980 (Figure III.), but more especially in R50540 (Figures II. and XI.), but these tumours do not give anything like the varied appearances seen in R47804 and R47504.

The possibility of these cells of sebaceous type being capable of interpretation as elements of unpigmented moles is mentioned in the main text.

CYST OF THE DUODENUM SIMULATING PYLORIC OBSTRUCTION.

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THE following notes should be of interest because of the rarity of the case and the interesting differential diagnosis which it introduces.

Clinical History.

E.F., aged three months, was admitted to the Royal Alexandra Hospital for Children, Sydney, on October 17, 1926, under Dr. E. H. M. Stephen. The only previous illness was bronchitis. The child weighed 3,713 grammes (eight pounds three ounces) at birth and on admission weighed 4,762·3 grammes (ten pounds eight ounces). From the beginning the child did not increase in weight at a normal rate and when six weeks old commenced to vomit about once daily, sometimes twice, the act being definitely projectile in character. The bowels were not constipated and the child appeared to be passing a reasonable amount of urine. For one day prior to admission it vomited every feeding.

In appearance the child was pale and wasted with a wizened shrunken face and limbs. The skin was moist, but toneless. Its abdomen was definitely full in its upper half and flattened in the lower. Distended veins were visible running vertically up from the level of the umbilicus towards the thorax. Wonderfully distinct, rhythmically recurring peristaltic waves followed each other from left to right across the epigastrium. In the region of the pylorus and below a large firm roughly rectangular mass was palpable about 6·25 by 4·3 centimetres (two and a half by one and three-quarter inches) which was quite freely movable in a vertical direction and could be pushed into the right loin or right iliac fossa. It felt hard and rather nodular and was apparently not at all tender. There were no enlarged glands to be felt anywhere. The liver and spleen were not enlarged and the remainder of the physical examination revealed no abnormality.

Figure Showing Specimen Removed at Autopsy.

Next day the baby was worse. It had vomited in a projectile fashion all its fluid none of which was bile-stained and some vomits were of large volume. After taking thirty cubic centimetres (one ounce) of milk every hour for twenty-four hours without vomiting, it suddenly returned approximately the whole quantity very little changed. Stomach lavage and atropine failed to aid the child in retaining anything and the child was given small amounts of fluid *per rectum* which it retained.

It was operated upon by Dr. R. B. Wade next day. He found the pylorus to be quite normal, but the first part of the duodenum ended in a large fusiform tumour about the size of a goose egg which appeared cystic. The lower pole of the mass emitted the attenuated third part of the duodenum. The remainder of the small intestine was collapsed. Dr. Wade attempted to perform a posterior gastro-enterostomy, but the condition of the child became too bad for him to proceed. The patient died about sixteen hours later.

Post Mortem Findings.

The autopsy showed the stomach to be very dilated with very thick walls, particularly the pyloric antrum. The remainder of the abdominal viscera were perfectly normal except the duodenum, the second part of which was tremendously dilated with the longitudinal muscle fibres spread out over it by splitting. The mass was elongated, round and cystic, with the ascending colon lying on its anterior aspect and the kidney behind.

It was excised with its connexions, hardened in formalin and dissected. The mass was found to be due to a large cyst with thin fibrous walls which projected into the lumen of the duodenum and completely blocked it, causing its extreme distension. The cyst contents were clear and colourless with the following chemical characters. It contained a few scattered leucocytes, many epithelial cells and abundant mucin. There was no evidence of any tryptic ferment, but this may have disappeared.

The cyst was adherent to the duodenal walls about the papilla of Vater and appeared, as shown in the accompanying figure, to be suspended by the pancreatic duct which appeared to end blindly in its wall. The relation of the cyst to the duct resembled closely that of an apple to its stalk. The bile duct did not appear to be interfered with, as the child showed no signs of jaundice.

Microscopically the walls of the cyst were composed of three layers, from within outwards: (i) A layer of poorly developed columnar epithelium with a few unicellular mucous glands apparently analogous to the epithelium of the pancreatic duct, (ii) a layer containing sparse and scattered plain muscle fibres with no attempt made to group them longitudinally and circularly, (iii) a fibrous adventitious coat.

Comment.

The close resemblance of the symptomatology to that of pyloric obstruction was interesting, and also the speculation as to the nature of the mass. A cystic kidney, new growth of the pylorus or bowel and retroperitoneal tumours were all considered. Certain cases of incomplete occlusion of the duodenum can cause a swelling of similar character in the same situation.[1]

Unfortunately the condition of the child did not permit of a radiographic examination.

I can find no reference in the standard English literature to an exactly similar condition. Possibly the cyst was of congenital origin, arising from a perversion of the vacuolation which precedes the formation of a continuous lumen to the primitive entodermal tube. Certain mesenteric cysts can arise in this way.[2] If this was the case, the cyst grew towards the lumen instead of towards the mesentery, carrying with it the pancreatic duct and apparently occluding it and ultimately the duodenum itself.

The microscopical characters, particularly of the inner wall, however, suggest an origin from the pancreatic duct. It may even represent the greatly dilated duct of Santorini which could not be identified elsewhere in the neighbourhood.

Acknowledgment.

I wish to thank both Dr. R. B. Wade and Dr. E. H. M. Stephen for permission to publish the notes of this case and Dr. F. Tidswell for the excellent pathological examination.

References.

[1] "J. M. Smellie: "Three Cases of Congenital Duodenal Occlusion," *British Journal of Children's Diseases*, July, 1924, page 192.

[2] H. C. Cameron: "Some Forms of Vomiting in Infancy," *The British Medical Journal*, April, 1925, page 765.

The Medical Journal of Australia

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The Accessory Food Factors.

THE term vitamin implies a substance or series of substances which are essential to life. It is not a good term for many reasons. Notwithstanding the persistent attempts on the part of Funk and others, no one has isolated a chemical body endowed with physiological properties attributed to the so-called vitamins. The absence of one or other of the vitamins from dietary is not inconsistent with life, although some vitamin action is essential for growth and some is essential for healthy development. Accessory food factors would be a preferable term if it could be shown that the physiological effect is due to a chemical entity and not to a function of some complex molecule. In the absence of exact information it is permissible to postulate a series of substances and to attribute to the presence of these substances the physiological action known to follow the use of adequate diets. Unfortunately the full range of the accessory food factors has not yet been discovered. The knowledge possessed concerning the development of rickets as a result of a deficiency of fat soluble vitamin *A*, of beri-beri as a result of a deficiency of the antineuritic accessory food factor and of scurvy as a result of a deficiency of the water soluble factor *C* is of great importance. On the basis of this elementary knowledge a great deal of investigation in biochemistry and physiology has been carried out in recent years and an enormous literature has been created. Those who have given this subject the most careful attention, have realized that growth, development and the maintenance of health do not depend only on the intake of accessory food factors, but demand the ingestion of proteins, carbohydrates and fat, with water and salts as well. The layman speaks glibly of vitamins and the market is overstocked with foods reputed to be especially rich in these substances. But notwithstanding the popularity of the term and the alleged superabundance of foods containing these substances, very little accurate information is available for the benefit of the community. The medical profession has paid too little attention to the whole subject of dietary and is inclined to accept scraps of information as sufficient. It is astounding that the actual caloric value of many foods consumed in Australia has not been determined. Figures calculated for foods in Great Britain and in foreign countries have been accepted for the corresponding foods in Australia, although by analogy it has been shown that the food values vary considerably according to the source of the ingredients. This is particularly true of cereals and of meat. In recent years, however, many estimations have been made and in the near future we should have the results of a considerable number of analyses of foods produced in Australia.

While the estimation of heat units in the combustion of protein, carbohydrate and fat foods can be carried out in almost every chemical laboratory, the determination of the growth producing element of food demands special conditions. It is necessary to breed rats and mice under uniform conditions and to establish standards on which calculations may be made in connexion with foods undergoing examination. As far as we are aware such standard conditions do not exist at the present time in any laboratory in Australia. Preparations are being carried out in the Physiological Department of the University of Sydney for the establishment of a laboratory for the estimation of accessory food factors. As soon as the somewhat tedious preparatory work is completed, it is anticipated that some highly important work will be undertaken in the laboratory. If the plan matures, we shall have for the first time reliable information concerning the vitamin content of Australian meat, milk, bread, butter, cheese, eggs, vegetables and fruits. It may be contended that this information is not essential, in view of the fact that so much work has been done elsewhere. This argument is untenable, since the accessory food factor content varies widely in foods of the same class produced in different parts of the world. It would be strange indeed if this were not so. It is known for instance that the antirhachitic factor can be replaced by substances that have been exposed to ultra-violet radiation. If accessory food factors

are merely activators, it is possible that the growth-producing effects and the stimulation of calcium metabolism induced by ultra-violet rays may be similar to the vitamin action. No one supposes that the intensity of the ultra-violet radiation of sunlight is the same all over the world. We are justified in assuming that estimations of the vitamin values of various foodstuffs carried out in Australia will reveal some unexpected results. The establishment of such a laboratory will have a useful purpose not only for the standardization of foods in the several parts of the Commonwealth, but also for the better control of proprietary foods offered for sale in virtue of their reputed vitamin content. Some of these proprietary foods may prove of importance, particularly when they possess a high nutrient value combined with a good vitamin action. Others may be of low caloric value or of doubtful assimilability, in which case the accessory food factor content would be immaterial. Others again may fail in regard to the accessory food factor. The sooner this laboratory is started, the better it will be for Australia.

Current Comment.

PROGNOSIS IN ENDOMETRIAL CARCINOMA.

SUCH emphasis is laid by medical teachers on the necessity for investigating irregular uterine hæmorrhage at or about the menopause that there is no excuse for failure to discover malignant disease of the uterus in the presence of irregular manifestations. The diagnosis in cancer of the *corpus uteri* may be and generally is much more difficult than in cancer of the cervix. The cervix can be palpated and inspected whenever any question as to its involvement is raised. Excision of a piece of tissue for microscopical examination will generally lead to a decision. With the body of the uterus more difficulty is experienced. Palpation is possible and inspection is not possible. Frequently no enlargement is palpable and the unwary may be lulled into a false feeling of security. Even when diagnostic curettage is carried out, the pathologist is not infreqently unable to determine the nature of the pathological change, if any. Malignant disease of the body of the uterus may occur in one of several forms and these differ from one another in degrees of malignancy. It is thus obvious that recognition of type of tumour will have a considerable influence on prognosis and possibly on treatment. In other words it is not sufficient to make a diagnosis of malignant disease; investigation must be carried out until the type is recognized.

W. S. Lindsay has recently discussed the variations in the prognosis of endometrial carcinoma as indicated by the histological structure.[1] He holds that once the diagnosis of endometrial carcinoma has been made, little or no interest is taken in the histological finding. This disease is generally regarded and treated as one of uniform type. Hence there has developed an entirely false conception of carcinoma of the fundus and the more malignant forms are treated with a leniency unwarranted by the facts. This is undoubtedly correct, but at the same time other factors have to be considered. Radical removal of cancer of the uterus by such procedures as the Wertheim operation is accompanied by what must be regarded as an excessively high mortality rate. But few surgeons have acquired efficiency and dexterity in the performance of this operation and it cannot be doubted that their proficiency has been attained as a sequel to a high initial mortality rate. Many surgeons hold, especially in view of the present status of radiation therapy, that surgical procedures of so heroic a nature as the Wertheim operation are not justified, that the few lives saved from death by malignant disease are equalled by those lost from post-operative shock. This point of view may be responsible for some of the leniency to which Lindsay refers. Lindsay also holds that many patients with the more benign forms of growth are subjected to a more severe operation which would appear to be unnecessary. This is the other side of the picture and most surgeons will agree that, provided death from postoperative shock is reduced to a minimum, it is better to be too radical than to run the risk of leaving behind cancerous tissue. Although Ladinski has collected records of twenty-two cases in which undoubted cancer of the endometrium has been successfully removed by curettage only, very few surgeons would be content to rely on this method of treatment, even if they could feel satisfied that endometrium alone was involved.

The object of Lindsay's investigation has been to attempt by correlation of the pathological findings with the subsequent history to evolve a practical classification which will be of value both to the pathologist and to the clinician and which will be of prognostic value to the latter. For this purpose he used records of all patients suffering from fundus carcinoma, treated at the Memorial and New York Hospitals since 1917. Only those cases were omitted in which material for the histological examination was not available and in which a record of the patient's progress could not be obtained. Lindsay points out that histological examination of fundus carcinoma reveals a wide variation in structure and that it is possible to separate from the main mass of cases three groups which, though not very large, can in his opinion be looked upon as three distinct diseases. These are adeno-acanthoma, adeno-myocarcinomatosis and embryonal carcinoma. Adeno-acanthoma consists of a combination of glandular and epidermoid epithelium and it is held that the latter is derived by metaplasia from the superficial epithelium lining the body cavity. In adeno-

[1] *Surgery, Gynecology and Obstetrics*, May, 1927.

myocarcinomatosis the malignant process arises in a previously existing adeno-myomatosis. The histological diagnosis of this condition from scrapings removed by the curette is difficult and often impossible. The disease probably arises in foci of adenomyoma situated deep in the muscle wall; by the time it reaches the endometrium and gives rise to symptoms, the whole wall is permeated. In this condition a diagnosis of adeno-carcinoma is generally made. Embryonal carcinoma is a small but definite group. A tumour of this nature is composed of sheets of closely packed round and polyhedral cells which are small and contain large darkly-staining nuclei and little cytoplasm. The tumours included in these three groups form but a small percentage of those under discussion. The majority of malignant tumours of the body of the uterus have a definitely glandular structure. At one extreme are the almost completely differentiated types in which the carcinomatous nature is indicated by the increase of glandular tissue with diminution of the stroma, the increased and irregular size of the cells and the hyperchromatic nuclei. At the other extreme differentiation is slight and the tumour is composed of solid masses of atypical cells deeply infiltrating the supporting structures. Between these extremes is an unbroken series which Lindsay divides into two groups, *adenoma malignum* and adeno-carcinoma. *Adenoma malignum* includes those cases in which the general structure of the glandular elements is everywhere well maintained, so that a definite lumen appears, surrounded by a single or double layer of cells. Adeno-carcinoma includes those in which there is a definite loss of polarity and in which the stroma is infiltrated by solid cords or masses of cells.

The patients observed by Lindsay numbered seventy. Thirty-one have died. The period which had elapsed since treatment was undertaken, was short in many cases and this high mortality indicates that carcinoma of the fundus is not, as commonly supposed, of relatively low malignancy. It is obvious that the series of cases is too small to justify the drawing of general conclusions. Lindsay recognizes this. At the same time some important points are brought out by his observations. The three small groups, adeno-acanthoma, adeno-myocarcinomatosis and embryonic carcinoma were represented in the series by eight cases and the only fact to be learned is that the mortality is high. Six of the patients have died and the two living have survived operation for two years and seven months and two years and nine months respectively. The findings in *adenoma malignum* and adeno-carcinoma are striking; the former is much less fatal than the latter. Twenty-three patients fall into the *adenoma malignum* group. Twenty-one are alive and two have died. One of the latter was admitted to hospital for a recurrence following hysterectomy. Among the twenty-one patients living are twelve who are classified as "three-year-cures." Thirty patients fell into the adeno-carcinoma group. Of these twenty-one have died and nine are living. The nine living include four "three-year cures." The difference in the results obtained in these two is not explainable by the treatment adopted, for the same differences in result were found when the groups were compared according to treatment by radiation only, by hysterectomy only or by combined radiation and hysterectomy. Of ten patients with *adenoma malignum*, treated by radiation, only one has died. Of twenty-three with adeno-carcinoma, treated in a similar manner, seventeen have died. Of five patients with *adenoma malignum*, treated by hysterectomy only, one has died and two with adeno-carcinoma, treated in a similar manner, have died. Of seven with *adenoma malignum*, treated by radiation and hysterectomy, none have died and of five with adeno-carcinoma, treated in a similar manner, two have died. It is obvious that these figures are comparable only if the time which has elapsed since operation is similar in each group. The figures need not be given in detail in this place, but perusal of tables given by Lindsay reveals striking similarity in this regard.

The question of prognosis is inseparably bound up with that of the method of treatment. Hence as a result of consideration of these findings it will be seen that Lindsay is right when he refrains from giving any answer to the question as to whether the less malignant condition, *adenoma malignum*, may be controlled by less radical measures than those that are required for adeno-carcinoma. It is safe to conclude from his figures that a surgeon is justified in giving a more hopeful prognosis in *adenoma malignum* than in adeno-carcinoma. But the prognosis in either of these conditions will depend on the way in which treatment has been carried out and on the surgeon who has done the work. In *adenoma malignum* an operation of the Wertheim type will not be indicated, but in adeno-carcinoma the decision will depend on the experience, the judgement and the conscience of the operator. While it may not be desirable to subject every patient to surgical operation, but to subject some to radiation only, it should be emphasized that in the present state of knowledge operations should invariably be accompanied by radiotherapy If this is done, the differentiation between *adenoma malignum* and adeno-carcinoma, as suggested by Lindsay, will be helpful in prognosis. More work will have to be done on the three small groups described by Lindsay before any conclusion in regard to prognosis can be reached about them.

THE VISIT OF DR. KANAVEL AND PROFESSOR ELLIOT.

THE Melbourne Permanent Committee for Post-Graduate Work has made arrangements for two series of lectures to be delivered by Dr. Allen Kanavel and Professor Charles Elliot, of Chicago, United States of America, from August 23 to August 31, 1927. Members who can afford the time, will be well advised to attend the course and benefit by the personal teaching of these two eminent practitioners. Applications should be made as soon as possible.

Abstracts from Current Medical Literature.

SURGERY.

Surgery of the Spinal Cord and its Membranes.

DONALD ARMOUR (*The Lancet*, February 26, 1927) describes the recent advances in the surgery of the spinal cord and its membranes. Hippocrates, Fabricius and Ambroise Paré with others advocated the operative exposure of the vertebræ in fractures of the spine, but it remained for the younger Cline in 1814 to perform the first formal operation on the living. There followed much controversy on the subject and Brown-Séquard in 1870 supported the operation. Macewan had success in dissecting away fibrous tissue in cases of both fracture and in Pott's disease, while von Leyden, of Strassburg, in 1873 proposed the operation of laminectomy for spinal tumour. The first spinal tumour was removed by Horsley in 1887. Lumbar puncture was first used by Corning in 1885 and in 1913 Marie showed that there was a qualitative difference in the cerebro-spinal fluid above and below a spinal tumour. Cisternal puncture was first introduced as a clinical aid in diagnosis by Ayer, Wegeforth and Essick in 1919 and advocated as a means of reaching the subarachnoid space when the spinal subarachnoid space is blocked by tumour, adhesions or bony deformity; in combination with lumbar puncture as a means of irrigating and draining the spinal subarachnoid space; as the best method of administering remedies in meningitis. Ayer has elaborated a method of combined cisternal and lumbar puncture to discover spinal block, working on the knowledge that in the normal individual the cisternal and spinal fluids are identical, as also are the pressures and the oscillations due to coughing, sneezing, holding the breath and compressing the jugular veins. He states that protein increase is a more delicate indication of cord compression or meningeal involvement than is demonstrable block. Normally the total amount of protein in the cerebro-spinal fluid is approximately 25 milligrammes per one hundred cubic centimetres of fluid. In cases of block this figure may increase a hundredfold in the stagnant fluid below the block. In addition there also accumulates an excess of mineral salts and glucose and the venous engorgement causes the fluid to approximate in chemical composition to blood plasma. Xanthochromia and spontaneous coagulation may also occur. These features are sometimes known as the loculation or Froin's syndrome. Pneumomyelography was introduced by Dandy, of Baltimore. The patient is placed recumbent at an angle of 50° or 60° to allow air to rise rapidly to the subarachnoid space; ten cubic centimetres of fluid being removed by lumbar puncture, an equal quantity of air is injected. In the absence of spinal block the patient complains immediately of a fairly sharp pain in the posterior, middle and then anterior parts of the head, sometimes vomiting and collapse occur. Absence of the pain means blockage and as much air as possible is injected so as to get a better radiographic picture. The air method for X ray photographs is unreliable and Sicard with Forestier, of Paris, have substituted "Lipiodol" or "Iodipin" as the substance to be injected. "Lipiodol" is injected by cisternal puncture and gravitates down to the block. Cerebro-spinal fluid is not withdrawn and one to one and a half cubic centimetres of "Lipiodol" are injected. The patient is at once examined by X rays and the behaviour of the "Lipiodol" shadow watched and again in six hours and at the end of a week. In regard to risks it has been noted that there may be transient increase of root pains, soreness and stiffness of the back muscles, headache, rise of temperature and difficulty in micturition. The correct interpretation of the radiogram is of paramount importance. The evidence of the value of "Lipiodol" injection in localization is doubtful and the risks are not negligible. Painstaking ordinary methods of neurological examination are still the soundest and the best.

DONALD ARMOUR (*The Lancet*, April 2, 1927) in a Lettsonian Lecture on the surgery of the spinal cord and its membranes deals with the operative procedure for the relief of pain in inoperable malignant disease of the spine and in the crises of *tabes dorsalis* other than that afforded by ordinary therapeutic measures. For the relief of severe neuritic pains, division of the posterior nerve roots was first performed in 1888, since then it has been repeated with varying success. This operation was first performed for the lightning pains of tabes at Forster's request in 1908. Epidural and intradural injections of various substances have been tried with varying success. Various modifications of Forster's operation have been recommended and tried. Whilst in 1911 the general opinion was that Forster's operation held out the only prospect of radical cure of the lightning pains of *tabes dorsalis*, Frazier stated in 1917 that only 19% of patients could be regarded as cured, whilst the mortality of the operation was 15%. Leriche in 1922 stated that he had never seen a good result even after the most extensive section. The equivocal reports upon the results of posterior root section led Schuller to recommend in 1910 partial division of the spinal cord. Deliberate complete division of the spinal cord for intractable and excruciating pain had been suggested by Cushing in 1910 and was carried out by him and by the author in 1916. In 1911 Spiller recommended section of the antero-lateral tracts of the spinal cord in a case of malignant growth in the spinal cord. This operation was performed by Martin and the pain was considerably diminished. This same operation was performed by Souttar in a case of *tabes dorsalis* in 1915. This operation has been frequently performed and the results, especially in cases of pain due to inoperable malignant disease, have been on the whole very successful. After dealing with the anatomy of the cord and localization in the antero-lateral columns, the author describes the operation of section of the antero-lateral tracts. This should offer no special difficulties to the surgeon trained in neurological surgery. The tracts are divided to a depth of three millimetres. The success or failure of the operation depends entirely upon the accuracy with which it is performed. Peet says that the section should be performed about four segments above the segment to be rendered analgesic, whilst Frazier takes a slightly higher level. A new operation suggested by Godwin Greenfield for these conditions which obviates operation on both sides of the cord and minimizes the risk of injury to the pyramidal tract consists of a medial longitudinal section of the cord at the selected level. The main danger of this procedure is the risk of injuring the anterior spinal artery.

Primary Thyreoidectomy for Exophthalmic Goitre.

ARNOLD S. JACKSON (*Surgery, Gynecology and Obstetrics*, March, 1927) reports one hundred and twenty consecutive operations of primary thyreoidectomy for exophthalmic goitre with no deaths. He attributes the strides made in the surgical treatment of this disease in recent years to the improvement of operative technique, the introduction of the estimation of the basal metabolic rate and the use of iodine. In the preoperative treatment the basal metabolism is determined and certain printed instructions are given about diet, 0·09 gramme (one and a half grains) of "Luminal" is ordered to be taken every day and ten drops of Lugol's solution before meals and at bedtime. The patient returns in five days for a further examination. Only about one-third of the patients require hospital preparation. All patients are given thirty drops of the tincture of digitalis for three days before operation. All operations are carried out under scopolamine-morphine anæsthesia with "Novocaine." The preliminary use of iodine simplifies operation. The whole gland is removed with the exception of a small strip on the posterior border of each lateral lobe. The muscles are divided in practically all patients. After operation the administration of iodine one drop in four cubic centimetres (one fluid drachm) of water is started at once. Ten drops are given every half-hour for six doses and four thereafter until the following morning. Each day the dosage is increased by ten drops. To every five

hundred cubic centimetres of saline solution, given by the rectum, ten drops of Lugol's solution are added. Morphine is used freely.

Ramisection and Periarterial Sympathectomy in the Treatment of Spasticity.

M. LEMOINE (*La Presse Médicale*, March 30, 1927) reports considerable improvement in spasticity of both arms and the left leg following ramisection in a case of Parkinson's disease. He performed periarterial sympathectomy on the right leg with the same excellent result.

Operation for Traumatic Rupture of the Urethra in the Male.

FREDERIC A. BESLEY (*Surgery, Gynecology and Obstetrics*, March, 1927) describes a method of treating traumatic rupture of the urethra in the male which has been followed by rapid convalescence in six patients. Local or sacral anæsthesia may be employed. A sound is passed to the lacerated portion of the urethra and a long longitudinal incision made on to it in the mid-line of the perineum. The torn urethra is thoroughly exposed and a large rubber drainage tube is sutured into the wound down to the laceration. The wound is loosely packed with gauze which is removed next day and not replaced. The tube is removed in from six to seven days. Suture of the tear in the urethra is unnecessary. No catheter should be passed either through the tear into the bladder or through the urethra. Unless there has been extensive destruction of tissue, the patient voids urine by the urethra in ten days. If the patient does not void urine through the incision after operation or if any extravasation of urine occurs, suprapubic drainage may be required. The operation should be performed before any extravasation of urine occurs. After use of this method, plastic repair of the urethra will rarely be necessary.

Fracture of the Tibia and Fibula.

CHARLES E. FARR (*Surgery, Gynecology and Obstetrics*, January, 1927) describes four cases of fracture of both bones of the leg, in three of which the fracture of the tibia was compound. The author's treatment consisted in operation of the fibula alone. The operation is easy, simple and safe and gives fairly good results in fractures that would otherwise be extremely difficult to handle. The operation consists of cutting down on and exposing the fibular fracture. The ends of the fractured bone are approximated. When this is done, the fragments of the tibial fracture will be found in moderately good position.

Retrograde Appendicectomy, Miscalled "Sub-Serous."

F. M. CADENAT (*La Presse Médicale*, February 5, 1927) holds that in certain types of appendicitis in which the appendix is adherent, retrograde removal is better than the usual method of appendicectomy. Even by this method difficulty is sometimes experienced and in these circumstances "sub-serous" appendicectomy is indicated. This name "sub-serous" is a misnomer as the line of separation is in the sub-mucosa and not in the sub-serous layer. This has been confirmed by examination of appendices removed by this method. The method is indicated particularly when the appendix is adherent in either the pelvic or retro-caecal positions and is bloodless. The only contraindication is failure to find the base of the appendix. The danger is that of breaking the appendix. To prevent this the cut edges are grasped with fine artery forceps and the tissue between is divided in the same way as in division of a paraphimosis.

Prognosis in Rectal Cancer.

LOCKHART MUMMERY (*The Lancet*, December 25, 1926) deals with the prognosis in rectal cancer. Age is an important factor. In young people the disease tends to grow rapidly and quickly disseminates, in later life it is extremely chronic and slow-growing. The author doubts whether it is worth while operating at all on patients under thirty as all these patients die from prompt recurrence, no matter how drastic the operation or other treatment has been. The most important factor of all is the stage to which the disease has progressed when the patient is first seen. When an early diagnosis has been made and no invasion of surrounding tissues has occurred, the prognosis is good and becomes progressively worse as the disease advances. The prognosis also depends on the position of the growth. There are three main types, an epithelioma of the anus, an adeno-carcinoma of the rectum and an adeno-carcinoma of the recto-sigmoidal junction. In the two first the prognosis is good, if diagnosis is made early, in the third the prognosis is not so good as in growths of the rectum itself, owing both to the increased operative difficulty and the fact that the diagnosis is usually made later. Patients treated without operation between the ages of fifty-five and sixty-four have an average expectation of life of a little under two years. Epithelioma of the anus and cancer of the rectum are best dealt with by means of the perineal operation and a permanent colostomy, these being carried out in two stages; growths of the recto-sigmoidal junction are best treated by means of abdomino-perineal excision. Two hundred cases are reviewed. In the last hundred perineal resections on private patients the mortality was only 3%, in hospital patients it was higher. It is doubtful whether it is possible to reduce the mortality of abdomino-perineal resection below 25%. Over a five-year period the percentage of cures works out at about 53%. Further analysis shows that when an operation is undertaken at an early stage of the growth, the chances of cure on a five-year basis are three to one. When the muscular coat is involved or a large fixed growth is present, the chances over the same period are six to four against a cure. The disability caused by the operation and by the resulting permanent colostomy has been found not to be serious and many patients were able to follow their usual employment. The author discusses other methods of treatment. He holds that deep X ray therapy as a means of curing cancer of the rectum is a failure and it has been thought that patients treated by X rays in some instances died more rapidly from extensive secondary growths than would have been expected. He knows of no cure from the use of radium. An improved technique for the application of radium to these growths is described. Colloidal copper undoubtedly does good; two patients were apparently cured by this means. Colloidal copper is not dangerous, it delays the growth of the tumour and reduces the secondary ulceration, it prolongs the patient's life and increases his comfort and well-being. Treatment by colloidal lead is still in the experimental stage, the treatment is not without danger, but the results are encouraging.

Wiring and Electrolysis of Aneurysms.

HOBART A. HARE (*The Journal of the American Medical Association*, January 22, 1927) discusses the treatment of aneurysm by wiring and electrolysis. He draws his deductions from a series of thirty-six cases. He states that only sacculated aneurysms are suitable for wiring. The more sharply defined the sac, the better is the chance of a good result. This is especially true when the vessel itself is not dilated. This procedure is also suitable to the so-called false aneurysm if it is pulsating or growing, but is contraindicated if there is more than one aneurysm or if the sac is distal to a large fusiform formation of the vessel. If syphilis is present, it is doubtful whether iodides should be used after the operations, as they may prevent clotting. The best wire to use is platinum-gold-silver wire, silver wire in itself will not coil and if too much platinum is used in the wire, it will have too much spring. The wire should have only enough stiffness to enable the operator to push it through the electrolytic needle and should be wound on a small spool so that it will coil in the sac in small snarls. The needle should be thoroughly insulated so as not to destroy the tissues by electrolysis when it passes through them. The current used should be six or eight dry cells controlled by a rheostat, the amount of current never exceeding fifty milliampères. The most pronounced good result of the procedure is the almost complete amelioration of pain. The life of the patient is lengthened by the arrest of the growth of the aneurysm. The author warns operators against mistaking gummata of the chest wall with transmitted pulsations for aneurysm.

Public Health.

THE HEALTH ORGANIZATION OF THE LEAGUE OF NATIONS.

REFERENCE has frequently been made during the past few years in this journal to the Health Organization of the League of Nations. The reader may be in doubt as to the constitution of the Health Organization and as to its machinery and the scope of its activities. It is probable that this auxiliary instrument of the League of Nations may play a very prominent part in the development of hygiene in the future and it may function as the coordinator and mouthpiece of the world's nations in combined efforts to eliminate preventible diseases. In these circumstances it may be of interest if we publish a general account of the aims, objects and machinery of the Health Organization.

THE LEAGUE OF NATIONS.

The League of Nations is an international institution that uses as its chief weapons publicity and persuasion. It imposes no forced decisions, but seeks to bring about amicable understandings and coordinated efforts. It will be remembered that the League of Nations came into being in January, 1920. Its members agreed to the terms of its twenty-six articles as a means of promoting international cooperation and of achieving peace and security. Each member agrees not to resort to war, to maintain open, just and honourable relations with other nations, to submit to the principles of international law in regard to the conduct of governments toward one another and to maintain justice and scrupulous respect for all treaty obligations in the dealings of organized peoples with one another. The members of the League of Nations are States; consequently all action undertaken by it must be proposed by the Government of a State enjoying membership. The decision of the League of Nations to undertake any particular action implies that the majority of the members have approved of this action. The action thus becomes the combined effort of the constituent nations through the agency of their several representatives.

The League of Nations comprises the Assembly, the Council, the permanent secretariat, the Permanent Court of International Justice and the International Labour Organization. In addition the League of Nations has power to appoint auxiliary bodies to deal with matters of more or less technical nature. The Assembly comprises all the members of the League of Nations. It meets annually and at such other times as it or the Council may determine. Each member or State is represented in the Assembly by not more than three individuals, but it has one vote. It is recognized that it is too large and cumbersome a body to carry out detailed international work. It has power to deliberate on matters arising between nations, especially those whose continuance might endanger the peace of the world. It appoints committees to deal with constitutional and legal questions, with work of the technical organizations, with disarmament, with budget and questions of internal administration, with social questions and with political questions. The meetings of the Assembly and of its committees are held in public.

The Council of the League of Nations consists of four permanent and six non-permanent members. Each State represented has one delegate and one vote. The Council has power to coopt members under special conditions. It is unnecessary for the present purpose to describe the functions and powers of the Council.

The permanent secretariat is a substantial organization. Its members are appointed by the Secretary-General with the approval of the Council. There is a deputy secretary-general and two under-secretaries. Each section is under the control of a director. The members of the responsible staff number seventy-five, while the clerical staff comprises no less than four hundred persons. The secretariat has a wide sphere of activity. It serves the Assembly and the Council and carries out the administrative work for these bodies. It also acts within defined limits independently of the two bodies and for this purpose it comprises eleven sections. The sections deal with political matters, financial and economic matters, transit, "minorities and administration (Saar territory and Danzig)" mandates, disarmament, health, social matters including opium and traffic in women and children, intellectual cooperation and international bureaux, legal matters and information. There is further a Latin-America bureau. The office organization of the permanent secretariat is complex and elaborate. Special facilities are provided for translations, précis writing, publication and filing. Each section is self contained. The official secretarial work for the particular subject has to be carried out by the staff attached to each section and all arrangements and correspondence is handled in the same way.

The Health Organization.

The Health Organization comprises an advisory council, a health committee and a secretariat which is a section of the permanent secretariat of the League of Nations. The Health Organization acts in cooperation with *l'Office international d'Hygiène publique*. The committee of *l'Office international d'Hygiène publique* acts as the advisory council of the Health Organization. This committee has its *siège* in Paris. It considers and discusses any questions submitted to it by the Health Committee. It should be mentioned that *l'Office international d'Hygiène publique* is constituted as a body of representatives of various governments whose special function is to organize international health conventions and to establish uniform action among health departments of the nations of the world in regard to epidemic and other transmittable diseases. In practice it appears that the advisory council as such enters into the affairs of the Health Organization to a very slight extent. Instead of this the committee of *l'Office international d'Hygiène publique*, acting under its own powers and constitution, collaborates with the Health Committee in regard to many matters of common interest.

The Health Committee.

The Health Committee consists of sixteen members. The chairman of the advisory committee, who is consequently president of *l'Office international d'Hygiène publique*, six members appointed by the Council of the League of Nations and nine members appointed by *l'Office international d'Hygiène publique* comprise the Committee. The selection is so arranged that each State that has been admitted to permanent membership of the Council of the League of Nations, is represented on the Health Committee. In October, 1926, the committee of *l'Office international d'Hygiène publique* selected the following nine members: Sir George Buchanan (England), Dr. H. Carrière (Switzerland), Surgeon-General H. S. Cumming (United States of America), Dr. C. Hamel (Germany), Dr. N. M. Josephus Jitta (the Netherlands), Professor Ricardo Jorge (Portugal), Dr. A. Lutrario (Italy), Dr. M. Tsurumi (Japan), and Dr. L. Raynaud (Algeria). M. M. O. Velghe, President of *l'Office international d'Hygiène publique*, is Vice-President of the Committee. The Council of the League of Nations appointed six members who were also nominated by the committee of *l'Office international d'Hygiène publique*: Dr. A. Alfaro (Argentine), Professor Léon Bernard (France), Dr. Carlos Chagas (Brazil), Dr. Witold Chodzko (Poland), Dr. J. D. Graham (India) and Dr. T. Madsen (Denmark). The Council has power to appoint four health assessors to act as members of the Health Committee and in February, 1927, it appointed Professor Jean Cantacuzene (Roumania), Professor B. Nocht (Germany), Professor D. Ottolenghi (Italy) and Professor G. Pittaluga (Spain). It also appointed three expert assessors to take part in the work of the Health Committee in an advisory capacity: Professor Alice Hamilton (United States of America), Professor C. E. A. Winslow (United States of America) and Dr. J. H. L. Cumpston (Australia). It has been announced that the Japanese member of the Committee will propose the name of a fourth expert assessor within a short time.

The Health Committee acts as the advisory body of the Council and the Assembly of the League of Nations and

is required to consider all questions connected with hygiene and health that may be referred to it by the Council. It is responsible for the technical work of the Health Section of the permanent secretariat of the League of Nations. It has power to conduct inquiries, to appoint subcommittees and to enlist the services of any person on the subcommittees. It issues an annual report to the Advisory Council.

From the annual report of the Health Organization for the year 1926 it appears that the Health Committee has appointed the following subcommittees or commissions. The malaria commission comprises ten members. In 1924 it undertook study tours in Serbia, Croatia, Slovakia, Greece, Roumania, Bulgaria and the "Union of the Socialist Soviet Republics." In 1925 the ground covered included Egypt, Palestine and Syria. Later in the same year the commission visited Spain, while in 1926 it extended the studies into Sicily. Several reports on the progress of the work of preventing malaria have been published. A general report of the work and achievements of the commission in the European tours is in course of preparation. In addition to these travelling investigations, other researches have been conducted into the therapeutic action of cinchona and its alkaloids. Epidemiological work has been carried out by individual members of the commission. These include the question of the importance of river deltas in regard to the development of anopheline mosquitoes, the value of sanitary campaigns in malarious districts, the study of reclaimed marsh land in its relation to mosquito life and so forth. The important problem of the survival of malarial infection of mosquitoes during hibernation, even during severe winters, has been closely investigated. On the recommendation of the commission the Health Committee instituted courses in the study of malaria prevention in London, Paris and Hamburg. The collaboration of the International Health Board of the Rockefeller Foundation has rendered this scheme a valuable and practical one. Scholarships have been awarded to the practitioners attending these courses.

The permanent standards subcommittee or commission has hitherto undertaken the standardization of antitetanus and antidysentery sera. A new standard unit has been adopted internationally. Two international units are equivalent to one American unit. The commission has also endeavoured to devise a standard for tuberculins, but at present the commission holds that it would be premature to propose the adoption of international rules for this purpose. The commission has further attacked the problem of the standardization of "Insulin," pituitary extract, digitalis, strophanthus, "Neo-Salvarsan" and "Neo-Sulpharsenol" and cod liver oil. The last standard is suggested in connexion with its vitamin *A* content. The commission has recently added to the list the biological tests for syphilis and is working at the formulation of rules to promote uniformity in the various countries.

A commission of experts on trypanosomiasis was dispatched to Entebbe in Uganda in 1925 for the purpose of studying this disease. The scope of the inquiries included an investigation into the existence of human immunity to sleeping sickness, the nature of this immunity and the factor determining it, a study of the function of wild and domestic animals as hosts of the virus, the determination of the relationship between *Trypanosoma gambiensis* and *Trapanosoma rhodesiensi* and an investigation of the blood of the tsetse fly, particularly in regard to the precipitin reaction.

There is also an opium commission, working under the clauses of the Opium Convention of 1925. There is also a cancer commission. This commission has endeavoured to gain information on certain statistical aspects of cancer incidence and mortality and to ascertain the prophylactic value of good obstetrics and the early treatment of carcinoma of the uterus in reducing suffering and death from this disease. The subject of cancer is also being attacked from the point of view of anthropology, while the biochemical aspect is also being considered. A tuberculosis commission, a commission on training in public health and a commission on variola and vaccination have been in existence for some time. These commissions or subcommittees issue reports, sometimes of a general nature to cover a coordinated investigation, sometimes by a single member on a special inquiry or research.

The Health Organization of the League of Nations is further charged with the promotion of knowledge of hygiene and preventive medicine. Arrangements have been made for the exchange of personnel engaged in public health work. As an example the interchange tour inaugurated in 1926 may be cited. Members of the public health departments of fifteen cities with over one hundred thousand persons were brought to London and benefited by a series of conferences instituted by the British Ministry of Health. Small groups were then arranged and these groups visited the various health divisions in Greater London. The visiting members were enabled to study the methods adopted both centrally and in the municipalities. Finally there was a conference held at Geneva at which the reports of technical interest were presented. Other study tours are organized from time to time, while an experiment has been carried out by extending study tours to medical officers specializing in particular branches of hygiene, such as the prevention of tuberculosis, the prevention of venereal diseases, child welfare, disinfection and so forth.

Opium and Dangerous Drugs.

There is a section of the permanent secretariat devoted to the control of the opium traffic. It appears that in accordance with a resolution adopted by the first meeting of the Assembly in 1920 a body was formed called the Opium Advisory Committee. It consisted of one representative of the Governments of China, France, Germany, Great Britain, India, Japan, the Netherlands, Portugal, of the Kingdom of the Serbs, Croats and Slovenes, Siam and the United States of America. Three assessors were appointed. This Committee submits to the Assembly of the League of Nations before each plenary session a report on all matters relating to the execution of arrangements in connexion with the traffic in opium and other dangerous drugs. In the report the Secretary includes a summary of the progress made and explains the conditions under which the several resolutions have been adopted by the Committee. Annual reports are received from the Governments represented on the Committee and points of importance are discussed in the report. Since its foundation the Advisory Committee on Traffic in Opium and Other Dangerous Drugs possesses members representing Bolivia, Switzerland and Italy. It may be pointed out that the Advisory Committee is working at a grave disadvantage since one country, namely Turkey, which produces raw opium in large quantities, has not yet ratified the Opium Convention held at the Hague. Records are published of the very large quantities of opium and cocaine seized in India and elsewhere and of the shipments of opium from one country to another.

Traffic in Women and Children.

In 1921 a general conference was summoned by the League of Nations for the purpose of securing a common understanding between the several Governments in regard to the traffic in women and children. It was hoped that united action might be taken to combat this traffic. The Governments of Denmark, France, Great Britain, Italy, Japan, Poland, Roumania, Spain and Uruguay were represented to form an advisory committee. The Council of the League of Nations has appointed representatives of the International Bureau for the Suppression of the Traffic in Women and Children, of the women's international organizations, of the Federation of National Unions for the Protection of Girls, of the International Catholic Association for the Protection of Girls and of the Jewish Association for the Protection of Girls as assessors with all the rights of delegates except the right to vote. Later the United States of America has been invited to appoint a delegate to the Advisory Committee and has accepted the invitation. The Advisory Committee works in a manner similar to that indicated above for the other committees of the League of Nations.

Financial Arrangements of the Health Organization.

When the League of Nations was instituted it was agreed that the constituent member States would share the expenses involved in its various undertakings. The budget is under the control of the Assembly. During the first four years the annual amounts made available for all purposes was about £902,350 a year. Of this sum approximately a quarter of a million pounds sterling is allocated to the International Labour Organization. This body undertakes some hygienic work. In 1925 the total expenditure on health organization amounted to £61,226, of which £28,965 was derived from grants by the Rockefeller Foundation. The amount estimated for 1926 was slightly more than the amount spent in 1925, but that estimated for 1927 was approximately the same. The League of Nations thus provides about £33,000 a year for health organization, as compared with £29,000 contributed by the Rockefeller Foundation. From the foregoing it will be seen that the extent and scope of the work is great, even when the annual budget is taken into account.

Special Correspondence.

LONDON LETTER.

By Our Special Correspondent.

Post-Graduate Work.

With many Australian doctors the autumn months would appear to be a popular time for visiting England and in most cases there is the intention of combining pleasure with business. As far as pleasure is concerned there are few people who will refuse to admit that autumn in England can be (and often is) a most delightful time of year. The warmth of the summer months lingering through September and into the early days of October, greatly enhances the enjoyment of the countryside with its brown and yellow tints, while a tinge of chilliness in the early mornings is conducive to a sense of general well-being. Visitors to England need not fear to lack amusement and pleasure—whatever may be their tastes—but it will not come amiss perhaps if a few notes are made as to the opportunities for business, that is post-graduate medical work, to be found in the Old Country.

The opening of the medical year does not start, strictly speaking, until October. In that month will begin series of lectures under the auspices of the Royal College of Physicians and the Royal College of Surgeons, also lectures arranged by the University of London, the Medical Society of London, University College, King's College and most of the London hospitals.

At the hospitals, post-graduate teaching will be begun again after the lull of the vacation months and a variety of courses will be offered from September onwards, for the delectation of visiting post-graduates. First might be mentioned the work obtainable at the two post-graduate colleges in London: the North-East London Post-Graduate College (Prince of Wales's General Hospital) which contains two hundred beds, of which sixty-two are devoted to medical patients, eighty-six to surgical and the rest distributed among the special departments. Clinical lectures and demonstrations are given daily and special intensive courses are given three times a year. The hospital is recognized by the University of London as a place of post-graduate study for the M.D. and M.S., and by the University of Cambridge in respect of bacteriology for the D.P.H. and of the diploma for radiology.

The second post-graduate college is that at the West London Hospital, which contains two hundred and twenty beds and serves a large area in the west end of London. The instruction given is mainly clinical in character and takes the form of demonstrations on actual cases as far as possible. Lectures are given by members of the staff of the College and by extra-mural lecturers on special subjects.

Besides these two definitely post-graduate colleges, courses of instruction will be held in other London hospitals from September onwards, including the Royal Northern (general) Hospital where short courses useful to candidates for the M.R.C.P. are held, the National Hospital at which a special two months' course will be held in October and November, the National Hospital for Diseases of the Heart when a special all-day two-weeks' course will be organized and the Hospital for Sick Children, Great Ormond Street, at which the usual series of lectures will be delivered. In addition to these hospitals special courses under the auspices of the Fellowship of Medicine will be held as follows in September: at the Westminster Hospital in medicine, surgery and the special departments, occupying all day from September 19 to October 1, at the Queen's Hospital for Children, in diseases of children, occupying the whole of each day from September 12 to 24, at the Royal Westminster Ophthalmic Hospital, taking place every afternoon from September 12 to October 1, at the Bethlem Royal Hospital in psychological medicine on Tuesdays and Saturdays, September 6 to October 1 and at the Royal National Orthopædic Hospital, for the whole of each day during two weeks.

In addition to these courses during the autumn and winter session the Fellowship will arrange series of weekly lectures on medical and surgical subjects and also weekly clinical demonstration in medicine, in surgery and in ophthalmology. These demonstrations and lectures are open to all members of the medical profession and have hitherto proved most popular.

Fellowship of Medicine.

The Fellowship of Medicine held its annual general meeting in February. From the Honorary Secretaries' report it was shown that steady progress had been made, as was proved by the increasing number of post-graduates enrolled for the various courses organized. Among the countries represented by the post-graduates there were forty-two from Australia and seventeen from New Zealand.

From April to December some 1,138 letters were dealt with by the Fellowship office and although naturally the majority of these were from the British Isles, yet over two hundred were from overseas. A most successful series of formal lectures on emergencies in medicine and surgery was arranged during the winter months, the average attendance at which was between fifty and sixty practitioners. There is no fee charged for these lectures and all members of the medical profession are welcomed. Besides the formal lectures, the Fellowship arranges programmes of free clinical demonstrations in surgery and in ophthalmology and it may be of interest to mention that it has now been decided to include demonstrations in medicine also. These demonstrations are carried on throughout the year except during the vacation months. The remainder of the report dealt with the propaganda undertaken by the Fellowship during the year, some remarks concerning its monthly journal, concluding with the usual votes of thanks to those who had kindly assisted the Fellowship in its work.

Lister Centenary.

April 4, 5 and 6 were the days chosen in London for the Lister centenary celebrations. On April 4 in the morning the delegates were received by the King at Buckingham Palace and in the evening a reception was held at the Royal Society of Medicine, 1, Wimpole Street. As it is in the House of the Royal Society of Medicine that the Fellowship of Medicine is accommodated and where it is hoped that all post-graduates visiting the Mother country for medical study will take the first opportunity of calling if advice and assistance is desired, I propose to give a short account of the reception. Sir James Berry, the President of the Royal Society of Medicine, and Lady Berry received the guests at 8.30 p.m. and at 9 o'clock Sir St. Clair Thomson gave an address entitled "A House Surgeon's Memories." The address was most illuminating and dealt briefly with the life of Lister, of his success in Edinburgh and of the opposition he had to contend with on his appointment to King's College Hospital, London. It is of interest to recall that two of the

doctors who accompanied Lister from Edinburgh—Dr John Stewart and Sir William Watson Cheyne—were present at the reception. The address was illustrated. On Tuesday there was a reception of the delegates at the British Medical Association, dinner with the Merchant Taylors Company and a reception at the Royal College of Surgeons. On Wednesday morning a memorial service was held in Westminster Abbey. In the afternoon discourses were held at the Royal Society of Medicine on the debt of the sciences to a three-fold genius. Sir Charles Sherrington paid his tribute to Lister as a physiologist and spoke of his work with the microscope; Professor William Bullock dealt with Lister as a bacteriologist and Sir Berkeley Moynihan in speaking of Lister as a surgeon said that his work which had conferred immortality upon him, had done for the craft of surgery what John Hunter had done for the science of surgery. The celebration terminated with a conversazione at the rooms of the Royal Society.

In conclusion I quote the perfect portrait of Lister written in verse by W. E. Henley, who at one time was his patient in the Edinburgh Infirmary.

His brow spreads large and placid, and his eye
Is deep and bright, with steady looks that still
Soft hues of tranquil thought his face fulfil—
His face at once benign and proud and shy.
If envy scout, if ignorance deny
His faultless patience, his unyielding will,
Beautiful gentleness and splendid skill,
Innumerable gratitudes reply.
His wise, rare smile is sweet with certainties
And seems in all his patients to compel
Such love and faith as failure cannot quell.

Correspondence.

SNAKE BITE.

SIR: From letters *re* above, appearing in THE MEDICAL JOURNAL OF AUSTRALIA, there is a misconception of the mechanism of snake bite. An anecdote will illustrate: Some years ago, while practising in Ravenswood, I engaged two miners to deepen a well. After examining it, one of them reported two brown snakes in the bottom. I sent for an hotelkeeper, Mr. John Moran, the most fearless snake charmer I have seen. He descended the well and brought up the two snakes in a sugar bag. He asked me in return to extract the snake poison and experiment with it on goats which I subsequently did to the loss of the latter. The bag with the snakes wriggling in it was lying on the ground; with a short stick he pinned one through the bag, put his hand in and pulled out by the neck a five-foot brown snake. I directed him to turn its head upside down, the poison fangs being in the lower jaw. I slipped a watch glass into its mouth with a piece of rubber on the under side to squeeze on. Shortly after, I saw a large glistening drop form near the root of each fang; it fell on to the watch glass after a period of five to ten seconds. Note well that it did not slide down to the tip of the tooth, because the latter is slightly bowed inwards. After about another ten seconds, two more much smaller drops fell and after waiting a little longer to be sure, there was no more. I extracted the watch glass. As the novelists say, "the snake gnashed its teeth with rage" and by accident bit itself. I noticed a little blood in the mouth. Immediately after its head dropped. It was dead. I then examined the fangs at leisure. As stated, they are bowed in. They are provided with grooves on the inner aspect, very deep near the roots, very shallow at the middle and disappearing before reaching the tips which are rounded and sharp.

From the above it will be seen that there is no danger in snake bite, unless on the bare skin and with the latter holding on for at least five seconds, then the poison, if any, will be found on the skin on the side of the fang wounds towards where the teeth of the upper jaw have marked.

Scarification of the wound should not be done; the poison is on the skin and may be inoculated into the blood thereby. There is little chance of its being in the fang wound.

From the above it can be seen that there is very little danger from snake bite and then only in people who go partly naked and are too timid to chuck off the snake at once, that is children and the natives of India.

What should be done? Generally nothing, but give assurance and stimulants for fright, otherwise apply strong permanganate to the wound and surrounding skin. Improvize a tourniquet and give stimulants.

Two punctures about half an inch apart indicate a bite by a snake which is probably poisonous, though not always; a few with those fangs are not so. There may be in addition the regular neat slight marks of the teeth of the upper jaw or a bruise from them, but usually nothing is visible but the two fang punctures of the lower jaw.

Yours, etc.,

D. P. O'BRIEN.

Rockhampton, Queensland,
June 1, 1927.

Naval and Military.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette* Number 61, of June 2, 1927.

AUSTRALIAN MILITARY FORCES.

Army Headquarters.

Major (provisionally) M. J. Holmes, D.S.O., Australian Army Medical Corps, relinquishes the appointment of Director of Hygiene, 30th April, 1927.

First Military District.

Australian Army Medical Corps.

Captain B. L. Hart is transferred to the Australian Army Medical Corps Reserve, 17th February, 1927.

Second Military District.

Australian Army Medical Corps.

The provisional appointment of Captain T. M. Greenaway is confirmed.

Third Military District.

Australian Army Medical Corps.

The provisional appointment of Major M. J. Holmes, D.S.O., is terminated, 30th April, 1927, and he is transferred to the Unattached List and to be Captain, 1st May, 1927.

Australian Army Medical Corps Reserve.

Honorary Lieutenant M. J. Morris is transferred from the Australian Army Medical Corps Reserve, 6th Military District, 2nd May, 1927.

Fifth Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captains.—John Leonard Day, Hugh Matheson Burns, and William Walter Seed, 5th May, 1927.

To be Honorary Lieutenant.—Stafford Fraser Sutherland, 5th May, 1927.

Sixth Military District.

Australian Army Medical Corps Reserve.

Captain F. Howson is transferred from the Australian Army Medical Corps Reserve, 2nd Military District, 22nd April, 1927.

CORRIGENDUM.

We note with regret that a typographical error remained uncorrected in our issue of last week. The writer of a letter on "The Red Back Spider Bite" was Dr. Andrew Balfour, not Alfred Balfour.

Books Received.

LISTER AS I KNEW HIM, by John Rudd Leeson, M.D., C.M. (Edinburgh), M.R.C.S., J.P. (Middlesex), F.L.S., F.R.A.S.; 1927. London: Baillière, Tindall and Cox. Demy 8vo., pp. 250, with 7 plates. Price: 8s. 6d. net.

LISTER AND THE LISTER WARD IN THE ROYAL INFIRMARY OF GLASGOW: A CENTENARY CONTRIBUTION. Published by Jackson, Wylie and Company, Glasgow, 1927. Imperial 8vo., pp. 132, with illustrations. Price: 12s. 6d. net.

REMINISCENCES OF LISTER AND OF HIS WORK IN THE WARDS OF THE GLASGOW ROYAL INFIRMARY, 1860-1869, by Sir Hector Clare Cameron, C.B.E., LL.D., M.D., C.M., F.R.F.P.S.; 1927. Glasgow: Jackson, Wylie and Company. Royal 8vo., pp. 45, with illustrations. Price: 1s. net.

PRACTICAL GASTROSCOPY, by Jean Rachet, M.D.; Authorized Translation by Fred. F. Imianitoff, D.S.C., B.A., M.R.C.S.; 1927. London: Baillière, Tindall and Cox. Demy 8vo., pp. 158, with illustrations. Price: 15s. net.

MANAGEMENT OF THE SICK INFANT, by Langley Porter, B.S., M.D., M.R.C.S. (England), L.R.C.P. (London), and William E. Carter, M.D.; Third Revised Edition; 1927. St. Louis: The C. V. Mosby Company. Royal 8vo., pp. 726, with illustrations. Price: $8.50 net.

VENEREAL DISEASE: ITS PREVENTION, SYMPTOMS AND TREATMENT, by Hugh Wansey Bayly, M.C.; Third Edition; 1927. London: Faber and Gwyer, Limited. Demy 8vo., pp. 242, with illustrations. Price: 10s. 6d. net.

PHARMACOPŒIA OF THE ROYAL PRINCE ALFRED HOSPITAL; 1927. Sydney: Angus and Robertson, Limited. Imp. 32mo., pp. 85. Price: 4s. net.

TRANSFUSION OF BLOOD, by Henry M. Feinblatt, M.D.; 1926. New York: The Macmillan Company; Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 137, with illustrations. Price: 15s. net.

Diary for the Month.

JUNE 20.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 21.—Tasmanian Branch, B.M.A.: Council.
JUNE 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 22.—Victorian Branch, B.M.A.: Council.
JUNE 24.—Queensland Branch, B.M.A.: Council.
JUNE 25.—Eastern District Medical Association (Kempsey), New South Wales.
JUNE 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 28.—Illawarra Suburbs Medical Association, New South Wales.
JUNE 30.—New South Wales Branch, B.M.A.: Branch.
JUNE 30.—South Australian Branch, B.M.A.: Branch.
JULY 1.—Queensland Branch, B.M.A.: Branch.
JULY 5.—Tasmanian Branch, B.M.A.: Council.
JULY 5.—New South Wales Branch, B.M.A.: Council (Quarterly).

Medical Appointments.

Dr. Hugh Gilmour Wallace (B.M.A.) has been appointed to act as Medical Inspector of Seamen, Newcastle, New South Wales, for the purposes of the *Navigation Act* 1912-1916 during the absence at any time of Dr. P. W. Mitchell (B.M.A.) on leave or special duty.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xxii.

ADELAIDE HOSPITAL, SOUTH AUSTRALIA: Pathological Registrar.
MELBOURNE HOSPITAL, VICTORIA: Honorary Surgeon to Out-Patients.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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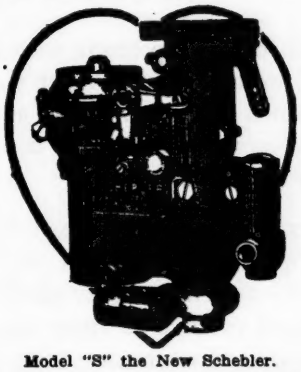

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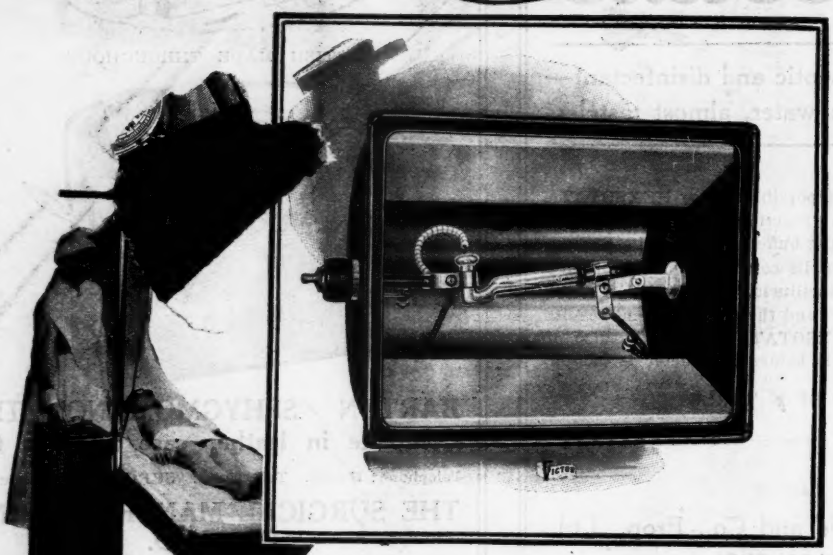

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